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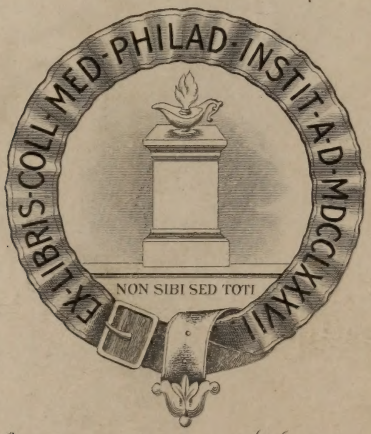

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TRANSACTIONS

OF THE

COLLEGE OF PHYSICIANS

OF

PHILADELPHIA

THIRD SERIES

VOLUME THE FORTY-FIFTH

PHILADELPHIA
PRINTED FOR THE COLLEGE
1923

NOTICE

The present volume of TRANSACTIONS contains the papers read before the College from January, 1923, to December, 1923, inclusive.

The Committee of Publication thinks it proper to say that the College holds itself in no way responsible for the statements, reasonings, or opinions set forth in the various papers published in its TRANSACTIONS.

EDITED BY

WALTER G. ELMER, M.D.

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1805	WILLIAM SHIPPEN
1809	ADAM KUHN
1818	THOMAS PARKE
1835	THOMAS C. JAMES[1]
1835	THOMAS T. HEWSON
1848	GEORGE B. WOOD
1879	W. S. W. RUSCHENBERGER
1883	ALFRED STILLÉ
1884	SAMUEL LEWIS[2]
1884	J. M. DA COSTA
1886	S. WEIR MITCHELL
1889	D. HAYES AGNEW
1892	S. WEIR MITCHELL
1895	J. M. DA COSTA
1898	JOHN ASHHURST, JR.
1900	W. W. KEEN
1902	HORATIO C WOOD
1904	ARTHUR V. MEIGS
1907	JAMES TYSON
1910	GEORGE E. DE SCHWEINITZ
1913	JAMES CORNELIUS WILSON
1916	RICHARD H. HARTE
1919	WILLIAM J. TAYLOR
1922	THOMAS R. NEILSON

[1] Died four months after his election.
[2] Resigned on account of ill-health.

COLLEGE OF PHYSICIANS OF PHILADELPHIA

1923

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FELLOWS

OF THE

COLLEGE OF PHYSICIANS OF PHILADELPHIA

DECEMBER, 1923

* Non-resident Fellows. † Fellows who have commuted dues. ‡ Deceased.

ELECTED

1892. Abbott, Alexander Crever, M.D., Sc.D., Dr. P.H., Professor of Hygiene and Bacteriology and Director of the School of Hygiene and Public Health; University of Pennsylvania. 4016 Pine St.

1921. Addison, William H. F., A.B., M.D., Professor of Normal Histology and Embryology in the University of Pennsylvania. Medical Laboratories, University of Pennsylvania.

1905. Adler, Lewis H., Jr., M.D., Formerly Professor of Diseases of the Rectum in the Graduate School of Medicine, University of Pennsylvania; formerly Prosector to the Professor of Anatomy in the University of Pennsylvania; Consulting Surgeon to the Charity Hospital. 313 S. Seventeenth St.

1914. Aiken, Thomas Gerald, M.D., Physician-in-Chief to the Chester County Hospital, West Chester Pa.; Pathologist to the Country Branch of the Rush Hospital for Tuberculosis. Berwyn, Pa.

1913. Alexander, Emory G., M.D., Surgeon to St. Christopher's Hospital for Children; Associate Surgeon to the Episcopal Hospital; Clinical Professor of Surgery in the Woman's Medical College of Pennsylvania, Demonstrator of Fracture Dressings in the Jefferson Medical College, and Assistant Surgeon to the Kensington Hospital for Women. 1701 Spruce St.

ELECTED

1906. Allen, Francis Olcott, Jr., A.B., M.D., Surgeon to the Presbyterian and Bryn Mawr Hospitals; Associate Surgeon to the Pennsylvania Hospital. 2216 Walnut St.

1896. Allyn, Herman B., A.B., M.D., Physician to the Philadelphia General Hospital. 3910 Chestnut St.

1888. Anders, James M., M.D., L.L.D., Professor of Medicine and Clinical Medicine in the Medico-Chirurgical College; Officer de l'Instruction Publique. 250 S. Seventeenth St.

1905. Anspach, Brooke M., M.D., Professor of Gynecology in the Jefferson Medical College; Attending Gynecologist, Jefferson Hospital; Gynecologist to the Stetson Hospital; Attending Gynecologist to the Bryn Mawr Hospital. 1827 Spruce St.

1905. Appleman, Leighton F., M.D., Demonstrator of Pharmacy and Materia Medica, and Instructor in Therapeutics in the Jefferson Medical College; Associate Professor of Ophthalmology, Graduate School of Medicine, University of Pennsylvania; Assistant Surgeon to the Wills Eye Hospital; Ophthalmologist to the Frederick Douglass Memorial Hospital and to the Burd School. 308 S. Sixteenth St.

1922. Arnett, John Hancock, A.B., M.D., Instructor in Medicine, Medical School, University of Pennsylvania; Assistant in Medicine and Students' Physician, University Hospital. 269 S. Twenty-first St.

1906. Ashhurst, Astley Paston Cooper, A.B., M.D., Professor of Clinical Surgery, University of Pennsylvania; Surgeon to the Episcopal Hospital and to the Philadelphia Orthopædic Hospital and Infirmary for Nervous Diseases. N. E. Cor. Sixteenth and Spruce Sts.

1893. Ashton, Thomas G., M.D., Physician to the Philadelphia General Hospital. Wynnewood, Pa.

1920. Astley, George Mason, M.D., Assistant Surgeon, Samaritan Hospital. 5317 Master St.

1914. Austin, J. Harold, B.S. (Univ. of Penna.), M.D., Professor of Research Med., University of Pennsylvania. Pepper Laboratory, University of Pennsylvania.

1906. Babbitt, James A., A.B. (Yale), A.M. (Haverford), M.D., Associate Professor of Oto-Laryngology, Graduate School of Medicine, University of Pennsylvania; Assistant Laryngologist and Aurist, Lankenau Hospital; Laryngologist

ELECTED

Mary Drexel Children's Hospital; Laryngologist and Aurist, Children's Hospital, Out-patient Department. 2117 Chestnut St.

1910. Baer, Benjamin F., Jr., B.S., M.D., Associate Professor of Ophthalmology, Graduate School of Medicine, University of Pennsylvania; Assistant Ophthalmologist, University Hospital; Consultant Ophthalmologist, Philadelphia Lying-in Charity Hospital. 1927 Spruce St.

†1892. Baker, George Fales, B.S., M.D. 1818 Spruce St.

1911. Baldwin, James Harvey, A.B., M.D., Surgeon to the Methodist Hospital. 1426 Pine St.

1889. Baldy, John Montgomery, M.D., Surgeon to the Gynecean Hospital; Consulting Surgeon to the Jewish and the Frederick Douglass Memorial Hospitals. Devon, Pa.

1916. Balentine, Percival L., M.D., Assistant Surgeon, Wills Eye Hospital; Demonstrator of Ophthalmic Surgery in the Philadelphia Polyclinic. 302 Weightman Building, 1524 Chestnut St.

1898. Balliet, Tilghman M., A.M., M.D., Professor of Therapeutics at Dartmouth College, Hanover, N. H.; Physician to the Old Man's Home. 3920 Sansom St.

1911. Barnard, Everett P., M.D., Obstetrician to the Maternity Hospital; Assistant Instructor in Obstetrics in the University of Pennsylvania. 245 S. Eighteenth St.

1921. Bauer, Edward L., M.D., Consultant Pediatrist, Germantown Hospital; Visiting Physician, Philadelphia Hospital for Contagious Diseases; Assistant Visiting Physician, Children's Hospital of the Mary J. Drexel Home. 6112 Germantown Ave.

1883. Baum, Charles, A.M., M.D., Ph.D. 1828 Wallace St.

1922. Bazett, Henry Cuthbert, M.A., M.D. (Oxon), F.R.C.S. (England), Professor of Physiology, University of Pennsylvania. Narbeth, Pa.

1908. Beardsley, Edward J. G., M.D., L.R.C.P. (Lond.), Associate Professor of Medicine in the Jefferson Medical College; Chief Clinical Assistant in the Out-patient Medical Department of the Jefferson Medical College Hospital; Assistant Physician to the Jefferson Hospital. 258 S. Sixteenth St.

*1916. Bell, William Hemphill, M.D., Captain, Medical Corps, U. S. Navy, Medical Officer of the Academy (Senior Medical Officer), U. S. Naval Academy, Annapolis, Maryland.

ELECTED

1923. Bergey, David H., A.M., M.D., B.S., Dr.P.H., Assistant Professor of Hygiene and Bacteriology, University of Pennsylvania. 206 S. Fifty-third St.

*1896. Beyea, Henry D., M.D. Ware Neck, Va.

*1903. Biggs, Montgomery H., M.D., Surgeon to the Rutherford Hospital; Chief Surgeon of the Carolina, Churchfield and Ohio Railway. Rutherfordton, N. C.

1918. Billings, Arthur E., M.D., Clinical Assistant in Surgery, Dept. B, Jefferson Medical College; Surgeon to Pennsylvania and Bryn Mawr Hospitals. 1703 Spruce St.

1917. Birdsall, Joseph C., A.B., A.M., M.D., Urologist, Polyclinic Section of the Medico-Chirurgical College and Hospital Graduate School of Medicine University of Pennsylvania; Assistant in Genito-urinary Dispensary, Presbyterian Hospital; Instructor in Surgery, University of Pennsylvania. 4610 Spruce St.

1908. Bland, Pascal Brooke, M.D., Chief Clinical Assistant in the Gynecological Department of the Jefferson Medical College Hospital; Instructor in Gynecology in the Jefferson Medical College; Gynecologist to St. Joseph's Hospital; Assistant Gynecologist to the Philadelphia General Hospital. 1621 Spruce St.

1917. Block, Frank B., A.B., M.D., Surgeon to the Jewish Hospital; Instructor in Gynecology in the University of Pennsylvania. 2035 Chestnut St.

1894. Bochroch, Max H., M.D., Neurologist to the St. Joseph's and Jewish Hospitals; Physician to the Psychopathic Wards of the Philadelphia General Hospital. 1539 Pine St.

1896. Boger, John A., A.M., M.D., Surgeon to St. Mary's Hospital; Surgeon to the Stetson Hospital; Surgeon to the Dispensary of the Episcopal Hospital. 2213 N. Broad St.

1923. Bond, Earl D., A.B., M.D., Physician-in-Chief, Pennsylvania Hospital, Department for Mental and Nervous Diseases; Professor of Psychiatry, Graduate School of Medicine, University of Pennsylvania. 4423 Larchwood Ave.

1911. Bonney, Charles W., A.B., M.D., Associate in Topographic and Applied Anatomy in the Jefferson Medical College. 1117 Spruce St.

1921. Borzell, Francis Frank, M.D., Roentgenologist, Frankford Hospital; Chief Clinical Assistant, Roentgen Department, Jefferson Medical College; *X*-Ray Consultant, State Hospital for the Insane, Norristown, Pa. 1933 Chestnut St.

ELECTED

1913. Boston, L. Napoleon, A.M., M.D., Professor of Physical Diagnosis in the Medico-Chirurgical College; Physician to the Philadelphia General Hospital; Pathologist to the Frankford Hospital. 2024 Chestnut St.

1919. Bowen, David Ralph, M.D., Radiologist to the Pennsylvania Hospital; Assistant Professor in Radiology, Graduate School of Medicine, University of Pennsylvania. 82 W. Lacrosse Ave., Lansdowne, Pa.

1923. Bower, John O., M.D. 2008 Walnut St.

1891. Boyd, George M., M.D., Professor of Obstetrics, Graduate School of Medicine, University of Pennsylvania; Obstetrician and Gynecologist to the Philadelphia Lying-in Charity; Obstetrician and Gynecologist to the Medico-Chirurgical Hospital. 1909 Spruce St.

1907. Boyer, Henry Percival, M.D., Neurologist to the Stetson Hospital; Physician to the Philadelphia Home for Incurables; Assistant Physican to the Orthopædic Hospital and Infirmary for Nervous Diseases. 4602 Baltimore Ave.

1907. Bradley, William N., Ph.G., M.D., Visiting Pediatrist to Howard Hospital; Visiting Pediatrist to Philadelphia General Hospital; Associate in Pediatrics, School of Medicine, University of Pennsylvania; Associate in Pediatrics, Graduate School of Medicine, University of Pennsylvania. 1725 Pine St.

1923. Braisted, William C., M.D., Ph.B., LL.D., D.Sc., F.R.C.S. (Edin.), Surgeon-General, U. S. Navy, retired; President, Philadelphia College of Pharmacy and Science. West Chester, Pa.

1921. Bransfield, John W., M.D., Visiting Surgeon to St. Agnes and St. Vincent's Hospital. 2025 Walnut St.

1903. Branson, Thomas F., M.D., Attending Physician to the Bryn Mawr Hospital. Rosemont, Pa.

1891. Brinton, Lewis, M.D., Physician-in-Chief to the American Hospital for Diseases of the Stomach. 1301 Medical Arts Bldg., Sixteenth and Walnut Sts.

1900. Brinton, Ward, A.M., M.D., Clinical Professor of Medicine, Woman's Medical College of Pennsylvania; Instructor in Medicine, University of Pennsylvania; Chief of Division of Tuberculosis, Department of Public Health of Philadelphia; Visiting Physician, Department for Tuberculosis, Philadelphia General Hospital. 1423 Spruce St.

ELECTED

1917. Bromer, Ralph S., A.B. (Yale University), M.D., Roentgenologist to the Episcopal and the Orthopædic Hospitals. 504 W. Mt. Airy Ave.

*1907. Brooks, Macy, A.B. (Princeton), M.D., Care Morgan, Harjes & Co., Paris, France.

1919. Brown, H. MacVeagh, M.D., Associate Physician, Presbyterian Hospital. 4608 Baltimore Ave.

1919. Brown, Henry P., Jr., M.D., Assistant Surgeon, Presbyterian and Pennsylvania Hospitals; Dispensary Surgeon, Children's Hospital. 2134 Spruce St.

1916. Brown, Samuel Horton, M.D. 1901 Mt. Vernon St.

1887. Brubaker, Albert P., A.M., M.D., Professor of Physiology and Medical Jurisprudence in the Jefferson Medical College. 3426 Powelton Ave.

1916. Buckley, Albert Coulson, M.D., Medical Superintendent, Friends' Hospital, Frankford; Associate Professor of Psychiatry, Graduate School of Medicine, University of Pennsylvania; Alienist to the Orthopædic Hospital and Infirmary for Nervous Diseases. Frankford, Phila.

1906. Burns, Stillwell C., M.D., Associate Professor of Surgery in the Graduate School of Medicine, University of Pennsylvania. 1925 Spring Garden St.

1892. Burr, Charles W., M.D., Professor of Mental Diseases in the University of Pennsylvania; Neurologist to the Philadelphia General Hospital. 1918 Spruce St.

1906. Butler, Ralph, M.D., Professor of Diseases of the Nose and Throat in the Philadelphia Polyclinic and the College for Graduates in Medicine; Chief of the Dispensary for Diseases of the Ear at the University Hospital; Assistant Professor of Otology in the University of Pennsylvania; Laryngologist and Aurist to the Lankenau Hospital. 2007 Chestnut St.

*1908. Cadbury, William W., A.M., M.D., Canton Christian College, Canton, China.

†1907. Cadwalader, Williams B., M.D., Assistant Professor of Neurology, School of Medicine, University of Pennsylvania; Neurologist to the Presbyterian Hospital; Consulting Neurologist to the Bryn Mawr Hospital. 1206 Medical Arts Building.

1905. Carmany, Harry S., M.D., Surgeon to Memorial Hospital, Roxborough; Associate Surgeon of the Episcopal

ELECTED

Hospital; Surgeon to the Dispensary of the Episcopal Hospital. 366 Green Lane, Roxborough.

1910. Carnett, John Berton, M.D., Professor of Surgery, Graduate School of Medicine, University of Pennsylvania; Associate in Surgery, Medical Department, University of Pennsylvania; Surgeon to Philadelphia General Hospital; Chief Surgeon to the American Stomach Hospital. 2012 Spruce St.

1905. Carpenter, Herbert B., M.D. 1805 Spruce St.

1895. Carpenter, John T., M.D., Attending Ophthalmologist to the Bryn Mawr Hospital. 2030 Chestnut St.

1917. Carson, John B., M.D., Physician to Episcopal Hospital; Assistant Physician, Pennsylvania Hospital. 1802 Pine Street.

1922. Case, Eugene A., M.D., Associate Professor of Pathology, University of Pennsylvania; Assistant Pathologist, Philadelphia General Hospital. Lansdowne, Pa.

1900. Chance, Burton K., M.D., Attending Surgeon to the Wills Eye Hospital; Consulting Ophthalmologist Eastern Pennsylvania Institution for the Feeble-minded and Epileptics; Ophthalmic Surgeon to the Pennsylvania Railroad Company. 1305 Spruce St.

1897. Cheston, Radcliffe, M.D., Visiting Physician to the Chestnut Hill Hospital; Consulting Physician to the Germantown Hospital, and to the Pennsylvania Institution for the Deaf and Dumb. Chestnut Hill.

1903. Chrystie, Walter, M.D. One of the Senior Physicians to Bryn Mawr Hospital. Bryn Mawr, Pa.

1899. Clark, John G., M.D., Professor of Gynecology in the University of Pennsylvania; Gynecologist-in-Chief to the University Hospital. 2017 Walnut St.

1896. Cleveland, Arthur H., M.D., Laryngologist to Pennsylvania Institution for Deaf and Dumb. 317 S. Fifteenth Street.

1910. Cloud, J. Howard, M.D., Assistant Physician to the Bryn Mawr Hospital; Attending Physician to the Children's House of Bryn Mawr Hospital. 11 W. Montgomery Ave., Ardmore, Pa.

1903. Coates, George Morrison, A.B., M.D., Professor of Otology, Graduate School of Medicine, University of Pennsylvania; Associate Surgeon, Ear, Throat, and Nose, Pennsylvania

ELECTED

Hospital; Consulting Laryngologist to the Philadelphia Orphanage and to the Sharon Hospital, Sharon, Conn. 1811 Spruce St.

1908. Codman, Charles A. E., M.D., Physician to the American Oncologic Hospital. 4116 Spruce St.

1922. Cohen, Leon Solis, A.B., M.D., Clinical Assistant of Roentgenology, Jefferson Medical College. 1923 Spruce St.

1907. Cohen, Myer Solis, A.B., M.D., Assistant Professor in Medicine, Graduate School of Medicine, University of Pennsylvania; Lecturer on Medical Jurisprudence, Woman's Medical College of Pennsylvania; Director of the Jules E. Mastbaum Research Laboratory of the Jewish Hospital; Pediatrist to the Jewish Hospital. 1833 Pine St.

1888. Cohen, Solomon Solis, M.D., Professor of Clinical Medicine in the Jefferson Medical College; Physician to the Jefferson Medical College Hospital, to the Philadelphia General Hospital, to the Jewish Hospital, and to the Rush Hospital. 1906 Walnut St.

1920. Cole, Charles J., A.B., M.D., Visiting Physician, Philadelphia Hospital for Contagious Diseases; Assistant Pediatrist, Polyclinic Hospital; Physician to Out-patient Department, Mary Drexel Home; Assistant Physician to Out-patient Department, Children's Hospital, University Hospital. 1521 Spruce St.

1898. Coles, Stricker, M.D., Assistant Professor of Obstetrics in the Jefferson Medical College; Assistant Obstetrician to the Jefferson and the Philadelphia General Hospitals; Visiting Physician to the Philadelphia Lying-in-Charity Hospital. Bryn Mawr, Pa.

*1901. Coley, Thomas Luther, A.B., M.D., Major, Medical Corps, U. S. Army. *Care*, Department Surgeon, Manila, P. I.

1903. Coplin, W. M. L., M.D., Professor of Pathology in the Jefferson Medical College; Pathologist to and Director of the Laboratories of the Jefferson Medical College Hospital; Pathologist to the Philadelphia General Hospital and to the Friends' Asylum for the Insane, Frankford; Bacteriologist to the Pennsylvania State Board of Health. Hotel Arlington, Atlantic City, N. J.

1912. Copp, Owen, A.B., M.D., Consultant for the Future Development of the Pennsylvania Hospital. 4401 Market St.

1911. Cornell, Walter Stewart, A.B., M.D., Director of Medical Inspection of Public Schools of the City of Philadelphia;

ELECTED

Chief of Medical Staff of the House of Detention; Lecturer on Osteology in the University of Pennsylvania. 1919 Cherry St.

1914. Corson, Edward Foulke, M.D., Associate in Dermatology, Jefferson Medical College; Assistant Dermatologist, Jefferson Hospital; Chief, Skin Dispensary, Jefferson Hospital; Dermatologist, Children's Hospital Dispensary. 1927 Spruce Street.

1907. Council, Malcolm S., M.D., Attending Physician to the Bryn Mawr Hospital; Attending Physician to the Cathcart Home at Devon. Bryn Mawr, Pa.

1904. Craig, Frank A., M.D., Instructor in Medicine in the University of Pennsylvania; Visiting Physician to the Henry Phipps Institute, University of Pennsylvania; Visiting Physician to the White Haven Sanatorium; Tuberculosis Consultant to the Presbyterian Hospital. 2011 Pine Street.

1907. Crampton, George S., M.D., Attending Surgeon to the Eye Department of the Pennsylvania Hospital and the Philadelphia Hospital for Contagious Diseases; Assistant Surgeon to the Wills Hospital; Lecturer on Physiologic Optics in the Philadelphia Polyclinic and School for Graduates in Medicine; Ophthalmologist to the Philadelphia Orphanage. 1819 Walnut St.

1920. Creighton, William J., B.A., M.D., Assistant Ophthalmic Surgeon, Germantown Hospital and Dispensary; Assistant Dispensary Surgeon, Department Ophthalmology, University Hospital; Instructor in Ophthalmology, University of Pennsylvania Medical School. 1905 Chestnut St.

1920. Cross, George Howard, M.D., Ophthalmologist to the Chester Hospital; Instructor in Ophthalmology, Graduate School of Medicine, University of Pennsylvania. Chester, Pa.

1917. Cross, Sumner H., A.B., M.D., Physician to the Abington Memorial Hospital, Abington, Pa. 412 York Road, Jenkintown, Pa.

1921. Crossan, Edward T., M.D., N. E. Cor. Sixteenth and Spruce Streets.

1904. Cruice, John M., M.D., Physician to the Henry Phipps Institute of the University of Pennsylvania; Physician to the Medical Dispensary of St. Agnes' Hospital; Instructor in Medicine in the University of Pennsylvania. 1932 Spruce St.

ELECTED

*1910. Cummins, W. Taylor, M.D., Pathologist to the Southern Pacific Hospital; Director of the Mary W. Harriman Research Laboratory, San Francisco, Cal.

1902. Currie, Charles A., M.D., Physician to the Germantown Hospital. West Walnut Lane, Germantown.

1896. Da Costa, John Chalmers, M.D., Professor of the Principles of Surgery and of Clinical Surgery in the Jefferson Medical College; Surgeon to the Philadelphia General and St. Joseph's Hospitals. 2045 Walnut St.

1887. Daland, Judson, M.D., Professor of Medicine, Graduate School of Medicine, University of Pennsylvania. 317 S. Eighteenth St.

‡1859. Darrach, James, M.D., Consulting Surgeon to the Germantown Hospital. Preston Apartments, Atlantic City, N. J.

1919. Davies, John R., Jr., M.D., Chief to the Out-patient Nose, Throat and Ear Department of the Pennsylvania, Bryn Mawr and Chestnut Hill Hospitals. 302 S. Nineteenth St.

1896. Davis, Charles N., M.D., Dermatologist to the Pennsylvania Hospital; Consulting Dermatologist to St. Agnes' Hospital; Assistant Physician to the Dispensary for Skin Diseases in the Howard Hospital. 1931 Spruce St.

1888. Davis, Edward P., A.M., M.D., Professor of Obstetrics in the Jefferson Medical College; Obstetrician to the Jefferson Hospital; Obstetrician and Gynecologist to the Philadelphia General Hospital; Consultant to the Preston Retreat. 250 S. Twenty-first St.

1916. Davis, Warren B., M.D., Rhinologist, Laryngologist, and Otologist, St. Agnes' Hospital; Instructor in Surgery, Jefferson Medical College; Instructor in Anatomy, Jefferson Medical College; Assistant Surgeon, Frankford Hospital. 135 S. Eighteenth St.

1900. Davisson, Alex. Heron, M.D. 4514 Springfield Ave.

1894. Deaver, Harry C., M.D., Professor of Surgery in the Woman's Medical College of Pennsylvania; Surgeon to the Episcopal Hospital and to the Children's Hospital of the Mary J. Drexel Home; Surgeon-in-Chief to the Kensington Hospital for Women. 1701 Spruce St.

1887. Deaver, John B., M.D., D.Sc., LL.D., John Rea Barton Professor of Surgery, University of Pennsylvania; Visiting Surgeon, Hospital of the University of Pennsylvania; Surgeon-in-Chief to the Lankenau Hospital. 1634 Walnut St.

ELECTED

1902. Dehoney, Howard, M.D. 240 S. Thirteenth St.

1923. De Long, Perce, M.D. 1909 Chestnut St.

1885. Dercum, Francis X., A.M., M.D., Ph.D., Professor of Nervous and Mental Diseases in the Jefferson Medical College; Consulting Neurologist to the Philadelphia General Hospital; Foreign Corresponding Member of the Neurological Society of Paris, and Corresponding Member of the Psychiatric and Neurological Society of Vienna. 1719 Walnut St.

1908. Despard, Duncan L., M.D., Surgeon to the Abington Memorial Hospital; Assistant Surgeon to the Jefferson Medical College Hospital; Demonstrator of Clinical Surgery in the Jefferson Medical College; Associate in Gynæcology in the Philadelphia Polyclinic Hospital. 256 S. Twenty-first St.

1912. Dewey, J. Hiland, Ph.B., M.D., Assistant Surgeon to Wills Eye Hospital; Ophthalmic Surgeon to St. Francis' Hospital, Trenton, N. J. 1436 Diamond St.

*1911. Dickson, Frank D., M.D. 1807 Federal Reserve Bank Building, Kansas City, Mo.

1908. Dillard, Henry K., Jr., M.D., Physician to the Out-patient Department of the Pennsylvania Hospital; Physician to the Dispensary of the Mary J. Drexel Home. 234 S. Twentieth St.

1920. Diven, John, A.B., M.D., Pediatrician to St. Christopher's Hospital and Babies' Hospital of Philadelphia. 2002 Spruce Street.

1920. Doane, Joseph Chapman, M.D., Superintendent and Medical Director, Philadelphia General Hospital, Assistant Professor and Instructor in Medicine; Graduate School of Medicine, University of Pennsylvania. Philadelphia General Hospital.

*1897. Dorland, W. A. Newman, A.M., M.D., Professor of Gynecology in the Post-graduate Medical School of Chicago; Professor of Obstetrics in the Chicago College of Medicine and Surgery; Visiting Obstetrician to Cook County Hospital. 25 E. Washington St., Chicago, Ill.

1907. Dorrance, George Morris, M.D., Surgeon to St. Agnes' Hospital; Demonstrator of Applied Anatomy in the Dental Department of the University of Pennsylvania. 2025 Walnut St.

1902. Downs, Robert N., Jr., M.D., Surgeon to the Dispensary of the Germantown Hospital. 6008 Greene St., Germantown.

ELECTED

1910. Drayton, William, Jr., M.D., Physician to the Philadelphia Hospital for Contagious Diseases; Physician to the Out-patient Department of the Pennsylvania Hospital; Physician to the Pennsylvania Institute for the Instruction of the Blind; Assistant Physician to the Philadelphia Orthopædic Hospital and Infirmary for Nervous Diseases. 1316 Locust St.

1911. Earnshaw, Henry Culp, M.S., M.D., Attending Physician to the Hospital of the Good Shepherd, Rosemont; Assistant Attending Physician to the Bryn Mawr Hospital; Attending Physician to the Bryn Mawr Children's Hospital; Pennsylvania Railroad Surgeon. Bryn Mawr, Pa.

*1887. Edwards, William A., M.D., Professor of Pediatrics in the Medical Department of the University of California. R. D. 2, Box 105, El Cajon, Calif.

1911. Eliason, Eldridge E., B.A., M.D., Associate in Surgery in the University of Pennsylvania; Surgeon to the Howard Hospital; Assistant Surgeon to the University of Pennsylvania Hospital, the Philadelphia General Hospital and the American Stomach Hospital. 330 S. Sixteenth St.

1904. Elmer, Walter G., B.S., M.D., Associate Professor of Orthopædic Surgery, Graduate School of Medicine, University of Pennsylvania; Associate Orthopædic Surgeon to the Polyclinic Hospital; Orthopædic Surgeon to the Jewish Hospital and to the Philadelphia General Hospital. 1801 Pine St.

1896. Ely, Thomas C., A.M., M.D. 3912 Chestnut St.

1901. Erck, Theodore A., M.D. 251 S. Thirteenth St.

1893. Eshner, Augustus A., M.D., Consulting Physician to Mercy Hospital. 1019 Spruce St.

*1905. Evans, Joseph S., Jr., A.B., M.D., Professor of Clinical Medicine in the University of Wisconsin; Consulting Physician, Madison General Hospital. 524 N. Francis St., Madison, Wis.

1905. Evans, William, M.D. 4007 Chestnut St.

1912. Eves, Curtis C., M.D., Aural and Laryngeal Surgeon to the Episcopal Hospital; Assistant in the Out-patient Department for Diseases of the Ear, Throat, and Nose of the Pennsylvania Hospital; Demonstrator of Operative Surgery of the Ear, Nose, and Throat in the Philadelphia Polyclinic. 247 S. Seventeenth St.

ELECTED

1894. Faries, Randolph, M.D. 2030 Chestnut St.

1922. Farley, David L., B.S., M.D., Instructor in Medicine, University of Pennsylvania; Assistant Physician, Pennsylvania Hospital. 1906 Pine St.

†1903. Farr, Clifford B., A.M., M.D., Director of Laboratories, Pennsylvania Hospital, Department for Mental and Nervous Diseases. Bryn Mawr, Pa.

1893. Farr, William W., M.D., Physician to the Leamy Home. 20 W. Ashmead Place, Chestnut Hill.

1884. Fenton, Thomas H., M.D., Ophthalmic Surgeon to St. Vincent's Home, the Baptist Home and the Widener Home for Crippled Children. 1319 Spruce St.

1907. Fetterolf, George, A.B., M.D., Sc.D., Assistant Laryngologist, University Hospital; Assistant Professor of Anatomy, University of Pennsylvania; Consulting Laryngologist, Kensington Hospital for Tuberculosis, Pennsylvania Institute for the Feeble Minded and Epileptic, and Eagleville Sanatorium. 2010 Spruce St.

1907. Fife, Charles A., A.B., M.D., Associate in Pediatrics in the University of Pennsylvania; Pediatrist to the Presbyterian Hospital; Physician to the St. Christopher's Hospital for Children; Assistant Physician to the Philadelphia General Hospital. 2104 Spruce St.

1884. Fisher, Henry M., M.D. 1027 Pine St.

1910. Fisher, John Monroe, M.D., Associate Professor of Gynecology in the Jefferson Medical College; Gynecologist to the Philadelphia, St. Agnes', and Pottstown Hospitals; Assistant Gynecologist to the Jefferson Medical College Hospital. 345 S. Nineteenth St.

1888. Flick, Lawrence F., M.D. 736 Pine St.

1916. Forst, John R., M.D. 166 W. Coulter St., Germantown.

1908. Foulkrod, Collin, M.D., Obstetrician to the Presbyterian Hospital; Assistant Demonstrator of Obstetrics in Jefferson Medical College. 3910 Chestnut St.

1908. Fox, Herbert, M.D., Director of the William Pepper Laboratory of Clinical Medicine, University of Pennsylvania; Pathologist to the Laboratory of Comparative Pathology of the Zoölogical Society of Philadelphia; Pathologist to the Rush Hospital; Pathologist to the Children's Hospital. 3902 Locust St.

†1885. Fox, Joseph M., M.D. Torresdale, Pa.

1906. Fraley, Frederick, Jr., A.B., M.D. Overbrook, Pa.

ELECTED

1903. Francine, Albert Philip, A.B., M.A., M.D., Clinical Director of Tuberculosis, U. S. Veterans' Bureau, Washington, D. C. 3309 Macomb St., N. W., Washington, D. C.

1897. Frazier, Charles H., A.B., M.D., Sc.D., John Rhea Barton Professor of Surgery, University of Pennsylvania. 3600 Walnut St.

1916. Funk, Elmer Hendricks, M.D., Assistant Professor of Medicine in the Jefferson Medical College; Medical Director and Physician-in-Charge of the Department for Diseases of the Chest of Jefferson Hospital; Assistant Physician to the Pennsylvania Hospital; Visiting Physician to the White Haven Sanatorium. 1318 Spruce St.

‡1910. Furbush, Charles Lincoln, M.D., Director of Public Health, Philadelphia. 4300 Spruce St.

1899. Gamble, Robert, G. M.D., one of the Attending Physicians to the Bryn Mawr Hospital. Haverford, Pa.

1912. Gaskill, Henry Kennedy, M.D., Assistant Professor of Dermatology in the Jefferson Medical College; Attending Dermatologist to the Philadelphia General Hospital. N. E. Cor. Sixteenth and Spruce Sts.

1917. Gerhard, Arthur Howell, M.D. 726 Richmond St.

1902. Ghriskey, Albert A., M.D. 3936 Walnut St.

1899. Gibbon, John H., M.D., Professor of Surgery in the Jefferson Medical College; Surgeon to the Pennsylvania Hospital and Consulting Surgeon to the Bryn Mawr Hospital. 1608 Spruce St.

1920. Gill, A. Bruce, A.B., M.D., Professor of Orthopædic Surgery, University of Pennsylvania; Surgeon to the Orthopædic Hospital; Orthopædic Surgeon to the Episcopal and Presbyterian Hospitals; Chief Surgeon to the Widener Memorial Industrial Training School for Crippled Children. The Lenox, Thirteenth and Spruce Sts.

*1913. Ginsburg, Nathaniel, M.D. 10 Peterboro Ave., Detroit, Mich.

1897. Girvin, John H., M.D., Gynecologist to the Presbyterian Hospital; Associate Professor of Gynecology Graduate School of Medicine, University of Pennsylvania. 2120 Walnut St.

1920. Githens, Thomas Stotesbury, M.D. Hamilton Court, Thirty-ninth and Chestnut Sts.

ELECTED

1906. Gittings, J. Claxton, M.D., Professor of Pediatrics, Graduate School of Medicine, University of Pennsylvania; Assistant Professor of Pediatrics, University of Pennsylvania, School of Medicine; Visiting Physician to the Children's Hospital of Philadelphia; Assistant Pediatric Physician, University Hospital. 1828 Pine St.

1905. Given, Ellis E. W., M.D., Surgeon to the Philadelphia Freemasons Memorial Hospital of the Masonic Home, Elizabethtown, Pa.; Surgeon to the Dispensary of the Episcopal Hospital. 2714 Columbia Ave.

1894. Gleason, E. B., S.B., M.D., LL.D., Professor of Otology in the Medico-Chirurgical College. 2033 Chestnut St.

1906. Goepp, R. Max, M.D., Professor of Medicine, Graduate School of Medicine, University of Pennsylvania. 1814 Spruce St.

1906. Goldberg, Harold G., M.D., Ophthalmic Surgeon to the Episcopal Hospital, Kensington Hospital for Women and Baltimore and Ohio Railroad. 1925 Chestnut St.

1908. Goodman, Edward H., M.D., Associate in Medicine in the University of Pennsylvania; Consultant to the Medical Dispensary of the University Hospital; Assistant Physician to the University Hospital; Assistant Physician to the Philadelphia General Hospital; Physician to the Presbyterian Hospital. 248 S. Twenty-first St.

1905. Gordon, Alfred, M.D., Neurologist to the Mt. Sinai, the Northwestern General, and the Douglass Memorial Hospitals. 1812 Spruce St.

1894. Graham, Edwin E., M.D., Professor of Pediatrics in the Jefferson Medical College; Pediatrist to the Jefferson and the Philadelphia General Hospitals; Physician to the Franklin Reformatory Home. 1713 Spruce St.

1904. Grayson, Charles P., M.D., Professor of Laryngology and Rhinology in the University of Pennsylvania; Physician-in-Charge of the Throat and Nose Department of the University Hospital; Otolaryngologist to the Philadelphia General Hospital. 262 S. Fifteenth St.

1910. Greenman, Milton J., M.D., Sc.D., Director of the Wistar Institute of Anatomy and Biology. Wistar Institute of Anatomy and Biology, Thirty-sixth St. and Woodland Ave.

1883. Griffith, J. P. Crozer, A.B., M.D., Ph.D., Professor of Pediatrics in the University of Pennsylvania; Corresponding Member of the Société de Pédiatrie de Paris; Physician to the Children's Hospital of Philadelphia. 1810 Spruce St.

ELECTED

1912. Griscom, J. Milton, B.S., M.D., Assistant Surgeon to the Wills Eye Hospital. 1925 Chestnut St.

1911. Gummey, Frank Bird, M.D., Visiting Physician to the Germantown Hospital and Dispensary; Visiting Physician to the Midnight Mission. 5418 Greene St., Germantown.

*1902. Gwyn, Norman B., M.D., Clinician in Medicine, Toronto General Hospital; Clinician in Medicine, Medical Faculty University of Toronto School. 48 Bloor St., E., Toronto, Canada.

1894. Hamill, Samuel McC., M.D., Professor of Diseases of Children in the Philadelphia Polyclinic and College for Graduates in Medicine; Pediatrist to the Presbyterian Hospital; Pediatrist to St. Vincent's Home. 1822 Spruce St.

1897. Hand, Alfred, Jr., M.D., Professor of Pediatrics, Graduate School of Medicine, University of Pennsylvania; Visiting Physician to the Children's Hospital, to the Children's Hospital of the Mary J. Drexel Home, and to the Methodist Hospital. 1724 Pine St.

1886. Hansell, Howard F., M.D., Professor of Ophthalmology in the Jefferson Medical College; Ophthalmic Surgeon to the Philadelphia General Hospital and to the Jefferson Medical College Hospital; Emeritus Professor of Diseases of the Eye in the Philadelphia Polyclinic. N. E. Cor. Seventeenth and Walnut Sts.

1889. Hare, Hobart A., LL.D., M.D., Professor of Therapeutics, Materia Medica, and Diagnosis in the Jefferson Medical College. 1801 Spruce St.

1903. Hart, Charles D., A.M., M.D., Inspector and Secretary of the Eastern State Penitentiary; National Executive Committee and Chairman of the Philadelphia Committee, Boy Scouts of America. Chestnut Hill.

1885. Harte, Richard H., M.D., (Hon.) F.R.C.S. Ireland, Adjunct Professor of Surgery in the University of Pennsylvania; Surgeon to the Pennsylvania and the Orthopædic Hospitals; Consulting Surgeon to St. Mary's, Memorial and the Bryn Mawr Hospitals. 1503 Spruce St.

1888. Hartzell, Milton B., A.M., M.D., LL.D., Professor of Dermatology in the University of Pennsylvania. Fayetteville, Pa.

1907. Hatfield, Charles James, A.B (Princeton), M.D., Executive Director of the Henry Phipps Institute for the Study,

ELECTED

Treatment, and Prevention of Tuberculosis; Visiting Physician to the White Haven Sanatorium. Phipps Institute, Seventh and Lombard Sts.

1872. Hays, I. Minis, M.D. 266 S. Twenty-first St.

1911. Heed, Charles R., M.D., Ophthalmologist to Girard College; Assistant Ophthalmologist to the Jefferson Medical College Hospital. 1205 Spruce St.

1908. Heineberg, Alfred, P.D., M.D., Associate in Gynecology in the Jefferson Medical College; Assistant Gynecologist to St. Agnes' Hospital. 1923 Spruce St.

1901. Heisler, John C., M.D., Professor of Anatomy, University of Pennsylvania. 3829 Walnut St.

1903. Henry, J. Norman, M.D., Physician to the Pennsylvania Hospital; Clinical Professor of Medicine in the Woman's Medical College of Pennsylvania; Assistant Physician to the Philadelphia General Hospital. 1906 Spruce St.

1891. Hewson, Addinell, A.B., A.M., M.D., Professor of Anatomy in the Philadelphia Polyclinic and College for Graduates in Medicine; Professor of Anatomy and Histology in the Temple University; Surgeon to Memorial Hospital, Roxborough. 326 S. Fifteenth St.

1909. Higbee, William S., M.D., President of the Pennsylvania State Board of Examiners for Registration of Nurses. 5121 Pine St.

1897. Hinkle, William M., M.D., Lecturer on the Anatomy and Physiology of the Vocal Organs in the National School of Elocution and Oratory. 923 N. Sixty-fourth St.

*1892. Hinsdale, Guy, A.M., M.D. Hot Springs, Virginia.

*1888. Hirsh, A. Bern, M.D., Chief of Physiotherapy and Occupational Therapy, United States Veterans' Bureau, District No. 2; Consulting Physician for Physiotherapy, Beth Israel Hospital. 71 W. Ninety-fourth St., New York.

1888. Hirst, Barton Cooke, A.B., M.D., LL.D., Professor of Obstetrics in the University of Pennsylvania; Gynecologist to the Philadelphia General and the Howard Hospitals. 1821 Spruce St.

1903. Hirst, John Cooke, M.D., Associate in Obstetrics, University of Pennsylvania; Obstetrician to St. Agnes' Hospital; Gynecologist and Obstetrician to the Philadelphia General Hospital; Gynecologist to Mt. Sinai Hospital. 1823 Pine St.

*1908. Hitchens, Arthur Parker, M.D., Instructor in Bacteriology, Army Medical School; Special Lecturer, Infection and

ELECTED

Immunity, Graduate School, Department of Agriculture; Advisory Professor in Bacteriology, American University. Washington, D. C.

1905. Hodge, Edward Blanchard, A.B., M.D., Surgeon to the Presbyterian and the Children's Hospitals; Associate Surgeon to the Pennsylvania Hospital; Associate Surgeon to the Widener Memorial Training School. 2019 Spruce St.

1913. Hoffman, Clarence, M.D., 1630 Pine St.

1906. Holloway, Thomas B., M.S., M.D., Vice-Dean for and Professor of Ophthalmology, Graduate School of Medicine, University of Pennsylvania, Instructor in Ophthalmology in the University of Pennsylvania; Attending Surgeon to the Wills Hospital; Ophthalmologist to the Pennsylvania Institution for the Instruction of the Blind at Overbrook. 1819 Chestnut St.

1914. Hooker, Richard S., M.D. 2147 N. Howard St.

1919. Hopkins, Arthur H., B.S. (Haverford), M.D., Associate in Medicine and Instructor in Practical Therapeutics, University of Pennsylvania; Physician to the Protestant Episcopal Hospital; Consulting Physician to the Chester County Hospital, West Chester, Pa. 1726 Pine St.

1908. Hoyt, Daniel M., M.D., Assistant Visiting Physician to the Philadelphia General Hospital. The Tracy, Thirty-sixth and Chestnut Sts.

*1912. Huber, G. Carl, M.D., Professor of Anatomy and Director of the Anatomic Laboratories in the University of Michigan. 1330 Hill St., Ann Arbor, Mich.

1892. Hughes, William E., M.D., Visiting Physician to the Philadelphia General Hospital; Consulting Physician to the Presbyterian Hospital. 3945 Chestnut St.

1923. Hume, John E., A.B., M.D., Visiting Physician, Medical Department, Howard Hospital. 1829 Spruce St.

1912. Hunter, John W., B.S., M.D. 4715 Chester Ave.

1921. Hunter, Robert J., M.D., Laryngologist Philadelphia General Hospital; Chief of Dispensary and Assistant Laryngologist and Aurist, Howard Hospital; Instructor in Otology, University of Pennsylvania, Graduate School. 928 N. Sixty-third Street.

1898. Hutchinson, James P., M.D., Surgeon to the Pennsylvania, the Methodist, the Children's, Memorial, and the Bryn Mawr Hospitals; Adjunct Professor of Surgery in the University of Pennsylvania. 133 S. Twenty-second St.

ELECTED

°1871. Ingham, James V., M.D. 1811 Walnut St.

1921. Ivy, Robert H., D.D.S., M.D., Professor of Clinical Maxillo-Facial Surgery, University of Pennsylvania; Oral Surgeon to the Hospitals of the Graduate School of Medicine, University of Pennsylvania and to the Philadelphia General Hospital; Consultant in Maxillo-Facial Surgery, Walter Reed General Hospital, Washington, D. C. 1503 Medical Arts Building.

1917. Jackson, Chevalier, M.D., Sc.D., Professor of Laryngology, Jefferson Medical College; Professor of Bronchoscopy and Esophagoscopy, Graduate School of Medicine, University of Pennsylvania. 128 S. Tenth St.

*1885. Jackson, Edward, A.M., M.D., Sc.D., Emeritus Professor of Ophthalmology in the University of Colorado; Emeritus Professor of Diseases of the Eye in the Philadelphia Polyclinic. 314 Fourteenth St., Denver, Col.

*1906. Jacobs, Francis Brinton, B.S., M.D., Assistant Surgeon to the Chester County Hospital. Whitford, Pa.

1922. Jameson, Howard Leon, M.D., Assistant Professor of Medicine, Graduate School of Medicine, University of Pennsylvania; Assistant Physician, Medico-Chirurgical and Polyclinic Hospitals. 2133 S. Sixteenth St.

1920. Janvier, G. Victor, M.D., Instructor in Obstetrics, University of Pennsylvania; Assistant Obstetrician to the Philadelphia General Hospital and the University of Pennsylvania Hospital; Associate in Gynecology at the American Stomach Hospital. Lansdowne, Pa.

1913. Jefferys, William Hamilton, A.B., A.M., M.D. New Street, Chestnut Hill.

1919. Jenks, Horace H., A.B., M.D., Medical Director, Associated Medical Clinics; Physician to the Out-Patient Department, Children's Hospital; Instructor in Pediatrics, Graduate School of Medicine, University of Pennsylvania. 918 Clinton St.

1898. Johnson, Russell H., A.B. (Princeton), M.D., Physician to the Pennsylvania Institution for the Deaf and Dumb. Chestnut Hill, Philadelphia.

1918. Jonas, Leon, M.D. 2253 N. 17th St.

1900. Jones, Charles James, A.M., M.D., LL.D., Ophthalmic Surgeon to St. Joseph's Hospital; Ophthalmic Surgeon to the House of the Good Shepherd, Germantown; Con-

ELECTED

sulting Ophthalmologist to St. Vincent's Home. 256 S. Fifteenth St.

1913. Jones, John F. X., B.S., A.B., A.M., M.D., Surgeon to St. Joseph's, Misericordia and St. Agnes' Hospitals. 103 S. Twenty-first St.

1900. Jopson, John H., M.D., Professor of Surgery, Graduate School of Medicine, University of Pennsylvania; Surgeon to the Presbyterian and the Children's Hospitals and Hospitals of the Graduate School of Medicine, University of Pennsylvania. 1824 Pine St.

1900. Judson, Charles F., A.B., M.D., Physician to Out-patient Department of the Lankenau Hospital and to the Southern Home for Children. 1005 Spruce St.

1902. Jump, Henry D., M.D., Assistant Physician to Philadelphia General Hospital; Physician to the Misericordia Hospital. 2019 Walnut St.

1886. Jurist, Louis, M.D. 1308 N. Broad St.

1903. Kalteyer, Frederick J., M.D., Associate Professor of Medicine, Jefferson Medical College; Assistant Physician, Jefferson Medical College Hospital; Consulting Physician to the Pottstown Hospital, Pottstown, Pa. 1530 Locust St.

*1910. Karsner, Howard T., M.D., Professor of Pathology in the Western Reserve University Medical School. Lakeside Hospital, Cleveland, Ohio.

1923. Kay, James, M.D., Visiting Physician, Episcopal Hospital; Assistant Instructor of Medicine, Jefferson Medical College. 1225 Spruce St.

1920. Keating, Peter McCall, M.D., Chief of Surgical Dispensary, St. Agnes Hospital; Assistant Attending Surgeon, Bryn Mawr Hospital; Assistant, Surgical Out-patient Department, Pennsylvania Hospital; Pathologist, Chester County Hospital. 2130 Pine St.

†1867. Keen, William W., M.D., LL.D., Sc.D. (Hon.) Ph.D. (Hon.), F.R.C.S. (Eng., Edin. and Ire.), Emeritus Professor of the Principles of Surgery and of Clinical Surgery in the Jefferson Medical College; Associé Etranger de l'Academie de Médecin, Paris; Honorary Member of the Société Belge de Chirurgie; Doctor honoris Causa of the University of Paris; Honorary Fellow Royal Society of Medicine, London, Honorary F.A.C.S. 1530 Spruce St.

ELECTED

1912. Keene, Floyd E., M.D., Instructor in Gynecology in the University of Pennsylvania; Assistant Gynecologist to the University Hospital; Gynecologist to the Chestnut Hill Hospital. Medical Arts Building.

1913. Kelly, Francis Joseph, M.D. 2035 Chestnut St.

*1887. Kelly, Howard A., A.B., M.D., LL.D. (Aberdeen, Wash. and Lee, and Univ. of Pa.), Professor of Gynecology in Johns Hopkins University and Gynecologist to the Johns Hopkins Hospital, Baltimore, Md.; Hon. Fellow of the Edinburgh Obstetrical Society, the Royal Academy of Medicine of Ireland, and of the Glasgow Obstetrical and Gynecological Society. 1418 Eutaw Place, Baltimore, Md.

1909. Kelly, James A., A.M., M.D., Visiting Surgeon to St. Mary's and Memorial Hospitals; Associate in Surgery in the Philadelphia Polyclinic and College for Graduates in Medicine; Assistant Visiting Surgeon to St. Joseph's Hospital. 1815 Spruce St.

1912. Kelly, Thomas C., A.M., M.D., Associate in Medicine, University of Pennsylvania; Assistant Professor of Medicine, Graduate School, University of Pennsylvania; Pediatrist to St. Christopher, Misericordia and St. Mary's Hospitals; Assistant Physician, Philadelphia General Hospital. 105 School Lane, Germantown.

1898. Kempton, Augustus F., M.D. 2118 Pine St.

1905. Kercher, Delno E., M.D. 1534 Pine St.

1921. Kern, Richard A., A.B., M.D., Associate in Medicine, University of Pennsylvania; Assistant Physician, University Hospital; Associate in Medicine, Graduate School of Medicine, University of Pennsylvania. 906 Medical Arts Building.

1920. Klauder, Joseph Victor, M.D., Associate Professor, Dermatology and Syphilology, Graduate School of Medicine, University of Pennsylvania; Professor of Dermatology, Woman's Medical College; Dermatologist, Misericordia, Woman's Medical College and American Stomach Hospitals; Assistant Dermatologist, Philadelphia General Hospital. 1807 Spruce St.

1920. Klein, Thomas, M.D., Associate Professor of Medicine, Graduate School, University of Pennsylvania; Assistant Physician, Presbyterian Hospital. 1717 Pine St.

1913. Klopp, Edward J., M.D., Instructor in Surgery in the Jefferson Medical College; Assistant Surgeon to the Germantown

ELECTED

Hospital; Chief Clinical Assistant in the Surgical Department of the Jefferson Hospital; Assistant Surgeon to the Out-patient Department of the Pennsylvania Hospital. 1611 Spruce St.

1895. Kneass, Samuel S., M.D., Associate in the William Pepper Laboratory of Clinical Medicine in the University of Pennsylvania. 1510 Walnut St.

1908. Knipe, Jay C., M.D., Ophthalmologist to the Jewish Hospital; Assistant Ophthalmologist to the Philadelphia General Hospital, and to the Mary J. Drexel Home; Chief of the Eye Clinic at the Jefferson Medical College Hospital; Demonstrator of Osteology and Syndesmology in the Jefferson Medical College. 2035 Chestnut St.

1919. Knipe, Norman Leslie, Ph.B. (Cornell); M.D., Instructor in Obstetrics, University of Pennsylvania; Assistant Obstetrician, University Hospital; Gynecologist, St. Mary's Hospital; Associate Gynecologist, American Stomach Hospital. 2018 Chestnut St.

1908. Knowles, Frank Crozer, M.D., Professor of Dermatology, Jefferson Medical College; Dermatologist to the Presbyterian and Children's Hospitals; Chief of Clinic, Pennsylvania Hospital. 2022 Spruce St.

1914. Kolmer, John A., M.D., Dr. P.H., M.Sc., Professor of Pathology and Bacteriology Graduate School of Medicine, University of Pennsylvania; Head of the Department of Pathology and Bacteriology in the Dermatological Research Laboratories of Philadelphia; Consulting Pathologist to St. Vincent's and Misericordia Hospitals; Consulting Serologist to Memorial and Germantown Hospitals. Cynwyd, Pa.

1904. Krauss, Frederick, M.D., Ophthalmic Surgeon to the Episcopal Hospital; Ophthalmic Surgeon to St. Christopher's Hospital for Children; Laryngologist to the Abington Hospital. 1701 Chestnut St.

1914. Krumbhaar, Edward B., A.B., Ph.D., M.D., Director of Laboratories, Philadelphia General Hospital; Associate Professor of Pathology, Graduate School of Medicine, University of Pennsylvania. Box 4310, Chestnut Hill.

1900. Krusen, Wilmer, M.D., Professor of Gynecology in the Medical Department of Temple University; Chief Gynecologist to the Samaritan and the Garretson Hospitals; Consulting Gynecologist to the Charity and Mercy Hospitals. 127 N. Twentieth St.

ELECTED

*1909. Laird, J. Packard, M.D. 34 N. Bayshore Drive, Miami, Florida.

1904. Landis, Henry R. M., M.D., Director of the Clinical and Sociological Departments of the Henry Phipps Institute of the University of Pennsylvania; Assistant Professor in Medicine in the University of Pennsylvania; Visiting Physician to the White Haven Sanatorium. 1817 Pine St.

1907. Langdon, H. Maxwell, M.D., Associate Professor of Ophthalmology, Graduate School of Medicine, University of Pennsylvania; Instructor of Ophthalmology, Under-Graduate School, University of Pennsylvania; Ophthalmologist of the Presbyterian, Orthopædic and Children's Hospitals. 1530 Locust St.

1922. Lawrance, Stuart J., M.D., Obstetrician, St. Mary's Hospital. 1332 Spruce St.

1922. Leach, Wilmon W., M.D., Physician to Dispensary, Children's Hospital. 2118 Spruce St.

‡1887. Leaman, Henry, M.D. Leaman Place, Pa.

1920. Leavitt, Frederick Headley, M.D. 1527 Pine St.

1904. Le Boutillier, Theodore, M.D., Professor of Pediatrics in the Woman's Medical College of Pennsylvania; Pediatrist to the Woman's College Hospital; Physician to the Philadelphia Hospital for Contagious Diseases. 2008 Walnut St.

1893. Le Conte, Robert G., A.B., M.D., Surgeon to the Pennsylvania Hospital; Consulting Surgeon to the Bryn Mawr and Germantown Hospitals. 2000 Spruce St.

1908. Lee, Walter Estell, M.D., Surgeon to the Germantown, Children's, Pennsylvania and Bryn Mawr Hospitals and the Associated Medical Clinics; Associate Professor of Surgery, Graduate School of Medicine, University of Pennsylvania; Consulting Surgeon, Henry Phipps Institute for Tuberculosis and the Medical Department of the State of Pennsylvania. 905 Pine St.

1903. Leffmann, Henry, A.M., M.D., D.D.S., Ph.D., Honorary Professor of Chemistry, Wagner Free Institute of Science; Emeritus Pathological Chemist, Jefferson Hospital; Lecturer on Research, Philadelphia College of Pharmacy and Science. 1839 N. Seventeenth St.

1892. Leidy, Joseph, M.D., Officier d'instruction publique, France; Consulting Physician to the Pennsylvania Training School for Feeble-minded Children. 1319 Locust St.

1920. Leopold, Simon S., M.D. 2025 Spruce St.

ELECTED

1915. Lewis, Fielding O., M.D., Associate in Laryngology in the Jefferson Medical College Hospital; Operating Clinical Chief of the Laryngological Dispensary and Clinical Assistant of the Otological Dispensary of the Jefferson Medical College Hospital; Laryngologist of the Philadelphia General Hospital. 261 S. Seventeenth St.

1877. Lewis, Morris J., M.D., Attending Physician to the Orthopædic Hospital and Infirmary for Nervous Diseases and Emeritus Physician to the Pennsylvania Hospital. 1316 Locust St.

1911. Lewis, Paul A., M.D., Director of the Pathological Department of the Henry Phipps Institute of the University of Pennsylvania; Professor of Experimental Pathology in the University of Pennsylvania. Henry Phipps Institute. Seventh and Lombard Sts.

1904. Lindauer, Eugene, M.D., Assistant Neurologist to the Philadelphia General and Polyclinic Hospitals; Instructor in Nervous Diseases, Graduate School of Medicine, University of Pennsylvania. 6451 N. Broad St.

1886. Lloyd, J. Hendrie, A.M. (Princeton), M.D., Neurologist to the Philadelphia General Hospital, and to the Methodist Episcopal Hospital; Consulting Neurologist to the State Asylum for the Chronic Insane at Wernersville, and to the Pennsylvania Training School for Feeble-minded Children at Elwyn. 4057 Spruce St.

1907. Lodholz, Edward, M.D., Isaac Ott Professor of Physiology, Graduate School of Medicine, University of Pennsylvania. Medical Laboratories, University of Pennsylvania.

1893. Longaker, Daniel, M.D., Obstetrician to the Kensington Hospital for Women and Visiting Obstetrician to the Jewish Maternity Hospital. 1806 Spruce St.

1907. Loux, Hiram R., M.D., Professor of Genito-urinary Surgery in the Jefferson Medical College; Surgeon to the Philadelphia General Hospital. Medical Arts Building, Sixteenth and Walnut Sts.

1921. Lucke, Baldwin, Dr. P.H., M.D., Assistant Pathologist, University and Philadelphia General Hospitals. Assistant Professor of Pathology, Medical School, University of Pennsylvania.

1923. Lueders, Charles W., A.B., M.D., Biochemist, Pennsylvania Hospital for Mental and Nervous Diseases; Research Worker, Gastro-enterology, Jefferson Medical College. Medical Arts Building.

ELECTED

1919. Lynch, Frank B., Jr., M.D., Pepper Laboratory, University of Pennsylvania.

1914. Lyon, B. B. Vincent, A.B. (Williams Coll.), M.D. (Johns Hopkins), Associate in Medicine Jefferson Medical College; Chief-of-Clinic, Gastro-Intestinal Department, Jefferson Hospital; Visiting Physician to Methodist Hospital. 328 S. Twenty-first St.

1900. McCarthy, Daniel J., M.D., Professor of Medical Jurisprudence (George B. Wood Foundation) in the University of Pennsylvania; Neurologist to the Philadelphia General and St. Agnes' Hospitals, and to the Henry Phipps Institute. 2025 Walnut St.

‡1903. McConnell, Guthrie, M.D., Pathologist to the Cleveland City Hospital; Associate in Pathology, Western Reserve Medical School. 2601 Hampshire Road, Cleveland, Ohio.

1913. McCrae, Thomas, B.A., M.D., F.R.C.P. (Lond.), Professor of Medicine in the Jefferson Medical College; Physician to the Jefferson and Pennsylvania Hospitals. 1929 Spruce Street.

1895. McFarland, Joseph, M.D., Sc.D., Professor of Pathology and Bacteriology in the Medical Department of the University of Pennsylvania; Pathologist to the Philadelphia General Hospital. 442 W. Stafford St., Germantown.

1913. McGlinn, John A., M.S., M.D., Associate Professor of Gynecology, Graduate School of Medicine, University of Pennsylvania; Gynecologist to St. Agnes' Hospital; Gynecologist, Philadelphia General Hospital. 1530 Locust St.

1905. McKenzie, R. Tait, M.D., LL.D., Professor of Physical Education and Director of the Department of Physical Education in the University of Pennsylvania. 2014 Pine St.

1916. McKnight, Howard A., A.B., M.D., Assistant Professor of Surgery, Graduate School of Medicine, University of Pennsylvania; Associate Professor of Surgery, Woman's Medical College of Pennsylvania; Surgeon, St. Mary's and Woman's College Hospitals; Chief, Surgical Clinic, Polyclinic and Medico-Chirurgical Hospitals. 345 S. Nineteenth Street.

1915. McLean, John D., M.D. 4316 Chestnut St.

*1900. McReynolds, Robert Phillips, M.D. 213 S. Broadway, Los Angeles, Calif.

ELECTED

1886. MacCoy, Alexander W., M.D., Consulting Laryngologist to the Bryn Mawr Hospital. Haverford Court, Haverford, Pa.

1910. Mackinney, William H., M.D., Assistant Surgeon to the Dispensary for Genito-urinary Diseases, University Hospital; Assistant in the Urological Dispensary of the Lankenau Hospital. 1701 Chestnut St.

1901. MacLeod, George I., M.D. Ardmore, Pa.

1914. Maier, F. Hurst, M.D., Associate in Gynecology to the Jefferson Medical College; Gynecologist to St. Joseph's Hospital; Gynecologist, Obstetrician and Consultant, Prenatal Clinic, Philadelphia General Hospital; Associate Gynecologist, St. Mary's Hospital. 2019 Walnut St.

1913. Major, C. Percy, M.D., Physician to the Dispensary of the Germantown Hospital; Pediatrist to the Dispensary of the Germantown Hospital; Pediatrist to the Abington Memorial Hospital. Tenth and Oak Lane.

1913. Manges, Willis F., M.D., Roentgenologist to the Jefferson Hospital; Director of the Roentgen Ray Laboratory in the Philadelphia General Hospital. 235 S. Fifteenth St.

1898. Marshall, George Morley, M.D., Laryngologist to the Philadelphia General Hospital; Laryngologist and Otologist to St. Joseph's Hospital. 1819 Spruce St.

1889. Martin, Edward, M.D., Surgeon to the University, Howard, Philadelphia General, and Bryn Mawr Hospitals. 135 S. Eighteenth St.

*1911. Meigs, Edward Browning, A.B., M.D., Physiologist in the Dairy Division of the United States Department of Agriculture. 1445 Rhode Island Ave., N. W., Washington, D. C.

1914. Mencke, J. Bernhard, A.B., M.D., Assistant Surgeon to the Out-patient Department of the Lankenau Hospital; Assistant Surgeon to the Stetson Hospital. 1816 Spruce Street.

1923. Mendelson, Walter L., M.D., 639 Church Lane, Germantown.

1914. Merrill, William Jackson, A.B., M.D., Associate Professor of Orthopædic Surgery, Graduate School of Medicine, University of Pennsylvania; Orthopædic Surgeon to the Children's Hospital, Germantown Hospital, Children's Seashore House; Consulting Orthopædic Surgeon to the Howard Hospital and Glen Mills School. 2017 Spruce St.

1923. Metheny, David G., M.D., 4609 Spruce St.

ELECTED

*1894. Miller, D. J. Milton, M.D., Associate Physician to the Children's Hospital, Philadelphia; Pediatrist to the Bamberger Home for Invalid Children, Longport, N. J. N. W. Cor. Pacific and California Aves., Atlantic City, N. J.

1922. Miller, Edwin B., M.D., Assistant Professor of Ophthalmology, Graduate School of Medicine, University of Pennsylvania; Associate in Ophthalmology, Temple University; Ophthalmologist, Roosevelt Hospital. 2008 Walnut St.

1910. Miller, Morris Booth, M.D., Professor of Surgery in the Philadelphia Polyclinic; Surgeon to the Douglass Memorial Hospital; Assistant Surgeon to the Philadelphia General Hospital. 2310 Pine St.

1921. Miller, T. Grier, A.B., M.D., Assistant Physician, University Hospital; Associate in Medicine, University of Pennsylvania. 110 S. Twentieth St.

1881. Mills, Charles K., M.D., LL.D., Emeritus Professor of Neurology in University of Pennsylvania; Neurologist to Philadelphia General Hospital; Consulting Physician to the Orthopædic Hospital and Infirmary for Nervous Diseases. 1909 Chestnut St.

1917. Mitchell, A. Graeme, M.D., Associate in Pediatrics, University of Pennsylvania; Assistant Pediatrist, Presbyterian Hospital; Clinical Assistant, Children's Hospital of Philadelphia; Physician to Dispensary Children's Hospital of Philadelphia. 1725 Pine St.

1904. Mitchell, Charles F., M.D., Surgeon to the Pennsylvania, Bryn Mawr, and Germantown Hospitals. 332 S. Fifteenth Street.

1921. Mohler, Henry K., P.D., M.D., Associate in Medicine, Jefferson Medical College; Medical Director, Jefferson Hospital. 319 S. Sixteenth St.

*1908. Montgomery, Charles M., A.B., M.D., U.S.P.H.S. Hospital, Oteen, N. C.

1882. Montgomery, Edward E., A.M., M.D., LL.D., Gynecologist to the Jefferson Medical College Hospital and to St. Joseph's Hospital. 1426 Spruce St.

1923. Moore, William F., M.D., Assistant, Bronchoscopic Clinic, Jefferson Hospital; Brochoscopist to Philadelphia General Hospital and Philadelphia Hospital for Contagious Diseases; Assistant Laryngologist, Children's Hospital, Out-Patient Department; Laryngologist to Tuberculosis Department, Philadelphia General Hospital. 255 S. Sixteenth St.

ELECTED

1918. Morgan, Arthur C., M.D., Professor of Applied Therapeutics, Medical Department, Temple University; Visiting Physician to Tuberculosis Department, Philadelphia General Hospital; Physician-in-Chief to the Frankford Hospital. 2018 Chestnut St.

1886. Morris, Caspar, M.D. 2050 Locust St.

1893. Morris, Elliston J., M.D., Physician to the Episcopal Hospital. 128 S. Eighteenth St.

1883. Morris, Henry, M.D., Emeritus Professor of Anatomy in the Woman's Medical College of Pennsylvania; Consulting Physician to St. Joseph's Hospital. 2310 Pine St.

‡1856. Morris, J. Cheston, M.D. "Fernbank," West Chester, Pa.

‡1906. Morrison, William H., M.D. 8021 Frankford Ave.

1897. Morton, Samuel W., M.D. 2007 Chestnut St.

1905. Müller, George P., M.D., Professor of Clinical Surgery, University of Pennsylvania; Surgeon to the University and Misericordia Hospitals; Consulting Surgeon to the Chester County and Chambersburg Hospitals. 1930 Spruce St.

1915. Musser, John H., Jr., B.S., M.D., Assistant Professor in Medicine in the University of Pennsylvania; Physician to the Philadelphia General Hospital; Physician-in-Charge, Medical Dispensary, University Hospital; Dispensary Chief and Assistant Physician in the Presbyterian Hospital. 262 S. Twenty-first St.

1905. Mutschler, Louis H., M.D., Surgeon to the Episcopal Hospital; Assistant Surgeon to the Orthopædic Hospital. 1625 Spruce St.

*1896. Myers, T. D., M.D. Hotel Cecil, 545 Post St., San Francisco, Calif.

1902. Nassau, Charles F., M.D., LL.D., Assistant Professor of Surgery in the Jefferson Medical College; Surgeon to St. Joseph's Hospital; Chief Surgeon to the Frankford Hospital; Surgeon to Mount Sinai Hospital. 1710 Locust St.

1886. Neff, Joseph S., A.M., M.D., LL.D., D.P.H., Narberth, Pa.

1887. Neilson, Thomas Rundle, A.M., M.D., Surgeon Emeritus to the Episcopal Hospital; Consulting Surgeon to St. Christopher's Hospital for Children; Emeritus Professor of Genito-urinary Surgery in the University of Pennsylvania. 1937 Chestnut St.

1905. Newcomet, William S., M.D., Physician in Charge of Radiological Department, Jefferson, Presbyterian and Oncologic Hospitals. 3501 Baring St.

ELECTED

1905. Newlin, Arthur, B.S., M.D., Physician to the Pennsylvania Hospital; Physician to the Dispensary of the Children's Hospital; Assistant Physician to the Orthopædic Hospital. 1804 Pine St.

1899. Nicholson, William Rufus, A.B., M.D., Professor of Gynecology, Graduate School of Medicine, University of Pennsylvania; Attending Gynecologist and Attending Obstetrician to the Methodist Episcopal Hospital; Attending Obstetrician to the Presbyterian Hospital. 2023 Spruce St.

1889. Noble, Charles P., M.D., Sc.D. (Hon.), Formerly Surgeon-in-Chief, Kensington Hospital for Women; Formerly Clinical Professor of Gynecology, Woman's Medical College. 1813 Spruce St.

1905. Norris, Charles C., M.D., Associate in Gynecology, University of Pennsylvania Medical School; Assistant Professor, Graduate School of Medicine, University of Pennsylvania; Assistant Gynecologist to the Hospital of the University of Pennsylvania; Gynecologist to the Children's Hospital, Philadelphia; Gynecologist and Obstetrician, Philadelphia General Hospital. Coronado, Twenty-second and Chestnut Streets.

1905. Norris, George William, A.B., M.D., Professor of Clinical Medicine in the University of Pennsylvania; Physician to the Pennsylvania Hospital. 1820 S. Rittenhouse Square.

*1901. Norris, Henry, M.D., Surgeon to the Rutherford Hospital. Rutherfordton, N. C.

1892. Norris, Richard C., M.D., Lecturer on Clinical and Operative Obstetrics in the University of Pennsylvania; Obstetrician in Charge of the Preston Retreat; Visiting Obstetrician to the Philadelphia General Hospital; Gynecologist to the Methodist Episcopal Hospital, and Consulting Obstetrician and Attending Gynecologist to the Southeastern Dispensary and Hospital. 500 N. Twentieth St.

1913. O'Neal, Alexander H., A.B., A.M., M.D., Physician to the Cathcart and Richardson Homes, Devon; Anesthetizer to the Bryn Mawr Hospital. St. Davids, Pa.

1920. Ostheimer, Alfred J., A.B., M.D., L.R.C.P. (London), M.R.C.S. (England), Neurologist to Abington Memorial Hospital; Chief, Neuro-psychiatric Section, District No. 3, United States Veteran's Bureau. Jenkintown, Pa.

ELECTED

1903. Ostheimer, Maurice, A.B., M.D., Assistant Professor of Pediatrics in the University of Pennsylvania; Physician-in-Charge of the Children's Dispensary and Assistant Visiting Physician, Children's Ward, University Hospital. 2214 De Lancey Place.

1913. Outerbridge, George W., A.B., M.D., Associate Professor of Gynecology, Graduate School of Medicine, University of Pennsylvania; Assistant Gynecologist to the Methodist Hospital; Gynecologist to the Abington Memorial Hospital. 1927 Spruce St.

1915. Owen, Hubley R., M.D., Surgeon to the Philadelphia General Hospital; Assistant Surgeon to the Orthopædic Hospital; Chief Surgeon of the Bureaus of Police and Fire, Philadelphia. 319 S. Sixteenth St.

1897. Packard, Francis R., M.D., Chief of the Ear, Nose and Throat Service, Pennsylvania Hospital; Consulting Laryngologist and Aurist, Bryn Mawr Hospital; Consulting Aurist Chestnut Hill Hospital; Laryngologist, Children's Hospital of Philadelphia. 304 S. Nineteenth St.

1898. Page, Henry F., M.D., Assistant Physician to the Lankenau Hospital and Physician to the Medical Dispensary of the same; Clinical Professor of Medicine in the Woman's Medical College of Pennsylvania. 315 S. Sixteenth St.

1906. Pancoast, Henry K., M.D., Professor of Roentgenology in the University of Pennsylvania and Roentgenologist to the University Hospital. Ardmore, Pa.

1909. Parish, Benjamin D., B.S., M.D., Assistant Instructor in Otology in the University of Pennsylvania; Assistant Surgeon to the Dispensary for Diseases of the Ear, University Hospital; Aurist and Laryngologist to St. Agnes' Hospital. 1927 Spruce St.

1899. Parke, William E., M.D., Consulting Obstetrician to the Episcopal Hospital; Associate Surgeon to the Kensington Hospital for Women; Gynecologist to the Frankford Hospital. 1739 N. Seventeenth St.

1910. Patterson, Ross Vernet, M.D., Dean and Associate Professor of Medicine, Jefferson Medical College; Physician to the Philadelphia General Hospital; Physician to the Protestant Episcopal Hospital; Assistant Physician to the Jefferson Hospital. 2126 Spruce St.

ELECTED

*1903. Pearce, Richard M., M.D., Sc.D., Director of Division of Medical Education, Rockefeller Foundation. 61 Broadway, New York, N. Y.

1922. Pelouze, P. S., M.D., Associate in Urology, University of Pennsylvania; Surgeon-in-Charge, Urological Dispensary, University Hospital. Medical Arts Building.

1909. Pemberton, Ralph, M.S., M.D., Physician to the Presbyterian Hospital; Director of the Department of Clinical Chemistry in the Pathological Laboratory of the Presbyterian Hospital. 2120 Sansom St.

†1889. Penrose, Charles Bingham, M.D., Ph.D. (Harvard), Formerly Professor of Gynecology in the University of Pennsylvania. 1720 Spruce St.

1914. Pepper, O. H. Perry, B.S., M.D., Assistant Professor of Medicine, University of Pennsylvania. Medical Arts Bldg.

†1902. Pepper, William, A.B., M.D., Dean of the School of Medicine, University of Pennsylvania; Member of the Council on Medical Education and Hospitals of the American Medical Association. University of Pennsylvania.

1916. Percival, Milton Fraser, M.D. 2332 S. Broad St.

1917. Peter, Luther C., A.M., M.D., Professor of Diseases of the Eye, Temple Medical School; Professor of Ophthalmology, University of Pennsylvania Graduate School. 1529 Spruce St.

1912. Petty, Orlando H., B.S., A.M., M.D., Professor of Diseases of Metabolism, Graduate School of Medicine, University of Pennsylvania; Chief of Department of Metabolism, Philadelphia General Hospital; Physician to Memorial Hospital; Chief of Department of Diabetes, Memorial Hospital. 1529 Pine St.

1905. Pfahler, George E., M.D., Professor of Roentgenology in the Medico-Chirurgical Graduate School of Medicine, University of Pennsylvania; Director of the *X*-ray Laboratories at the Howard Hospital, Polyclinic Hospital and the Misericordia Hospital; Honorary member, Scandinavian Radiological Society. 1321 Spruce St.

1915. Pfeiffer, Damon B., A.B., M.D., Assistant Professor of Surgery, Graduate School of Medicine, University of Pennsylvania; Surgeon, Methodist and Abington Memorial Hospitals; Assistant Surgeon, Presbyterian and Lankenau Hospitals. 1822 Pine St.

ELECTED

1907. Pfromm, George W., Ph.G., M.D., Assistant Physician to the American Stomach Hospital; Consulting Physician to the German Protestant Home for the Aged. 1431 N. Fifteenth St.

1907. Phillips, Horace, M.D., Assistant Surgeon U. S. Public Health Service; Examining Physician, Office of the District Attorney. 905 Land Title Building.

1883. Piersol, George A., M.D., Sc.D., Emeritus Professor of Anatomy in the University of Pennsylvania. 4724 Chester Ave.

1911. Piersol, George Morris, B.S., M.D., Professor of Medicine and Vice-Dean for Medicine, Graduate School of Medicine, University of Pennsylvania; Physician to the Philadelphia General and Methodist Episcopal Hospitals. 1913 Spruce St.

1917. Piper, Edmund Brown, B.S., M.D., Associate in Obstetrics, University of Pennsylvania Medical School; Assistant Professor in Obstetrics, University of Pennsylvania Graduate School; Assistant Obstetrician to the University Hospital; Assistant Obstetrician and Gynecologist to the Philadelphia General Hospital. 1936 Spruce St.

1905. Pitfield, Robert L., M.D., Pathologist to the Germantown Hospital; Bacteriologist to the Chestnut Hill Hospital for Lung Diseases. 5211 Wayne Ave.

1922. Pleasants, Henry, Jr., A.B., M.D., Assistant Visiting Physician, Chester County Hospital, West Chester, Pa.; Assistant in Neurological Clinic, Orthopædic Hospital. West Chester, Pa.

1896. Posey, William Campbell, M.D., Ophthalmic Surgeon to the Howard Hospital; Consulting Ophthalmic Surgeon to the Wills Eye Hospital; Chairman of the Commission on the Conservation of Vision for Pennsylvania. 2049 Chestnut Street.

1899. Potts, Charles S., M.D., Professor of Neurology, Graduate School of Medicine, University of Pennsylvania; Neurologist to the Philadelphia General Hospital; Consulting Neurologist, Lankenau Hospital; Consulting Neurologist, Hospital for the Insane, Atlantic County, New Jersey, 2018 Chestnut St.

*1907. Price, George E., M.D. Suite 620, Paulsen Bldg., Spokane, Wash.

1920. Quicksall, William E., M.D. 1819 Spruce St.

ELECTED

1908. Radcliffe, McCluney, A.M. (Lafayette), M.D., LL.D., Consulting Ophthalmologist, Presbyterian Hospital; attending Surgeon to the Wills Eye Hospital. 1906 Chestnut St.

1913. Randall, Alexander, A.M., M.D., Assistant Professor of Surgery in charge of the Department of Urology, University of Pennsylvania. 1306 Medical Arts Building.

1887. Randall, B. Alexander, M.A., M.D., Professor of Otology in the University of Pennsylvania; Ear Surgeon to the Children's Hospital; Consulting Aurist to the Pennsylvania Institution for the Deaf and Dumb, and to Memorial Hospital, Roxborough. 2009 Spruce St.

*1904. Ravenel, Mazyck P., M.D., Professor of Preventive Medicine and Bacteriology and Director of the Public Health Laboratory at the University of Missouri. 203 Hicks Ave., Columbia, Mo.

1920. Rehfuss, Martin E., M.D. N. E. Cor. 16th and Spruce Sts.

1921. Reiff, E. Paul, A.B., M.D., Member of Medical Staff, Methodist Episcopal Hospital. 5051 Chestnut St.

1919. Reiman, Stanley P., M.D. 516 Arbutus St., Germantown.

1923. Repplier, Sidney, J., M.D., Medical Director, Curtis Hospital, Curtis Publishing Company. Chestnut Hill, Phila.

1897. Rhein, John H. W., M.D., Professor of Nervous Diseases, Graduate School of Medicine, University of Pennsylvania; Neuropsychiatrist to the Philadelphia General Hospital; Consulting Neurologist to the Philadelphia Home for Incurables. 1732 Pine St.

1906. Rhein, Robert D., M.D., Chief Physician to the Clinic of the American Hospital for Diseases of the Stomach; Physician to the Philadelphia Home for Incurables; Examining Physician to the White Haven Sanatorium. S. W. Cor. 15th and Pine Sts.

1891. Rhoads, Edward G. M.D., 108 Queen Lane, Germantown.

*1910. Rhoads, Samuel, M.D. Pasadena, Calif.

1919. Richardson, Russell, A.M., M.D., Director of the Laboratory, Methodist Episcopal Hospital; Pathologist to Abington Memorial Hospital and the Associated Medical Clinics. 320 S. Sixteenth St.

1919. Ridpath, Robert F., M.D. 1928 Chestnut St.

1898. Riesman, David, M.D., Professor of Clinical Medicine, University of Pennsylvania; Visiting Physician to the Philadelphia General and University Hospitals; Consulting Physician to the Jewish and Woman's Hospitals. 1520 Spruce St.

ELECTED

1895. Ring, G. Oram, A.M., M.D., Orphthalmic Surgeon to the Episcopal Hospital; Ophthalmologist to the Widener Memorial Home for Crippled Children; Consulting Ophthalmologist to the American Oncologic Hospital. N. E. Cor. Seventeenth and Walnut Sts.

*1905. Risley, J. Norman, M.D. New Bedford, Mass.

*1878. Roberts, John B., A.M., M.D., Professor of Surgery, Graduate School of Medicine, University of Pennsylvania. 1520 Spruce St.

1899. Roberts, Walter, M.D., Professor of Otology in the Philadelphia Polyclinic; Otologist to the Methodist Episcopal Hospital; Laryngologist to the Philadelphia General Hospital. 1732 Spruce St.

1903. Robertson, William Egbert, M.D., Professor of Theory and Practice of Medicine and of Clinical Medicine in Temple University; Physician to the Episcopal, Samaritan, and Garretson Hospitals. 327 S. Seventeenth St.

1902. Robinson, James Weir, M.D. 326 S. Sixteenth St.

1903. Robinson, William Duffield, Ph.G., M.D. 2012 Mount Vernon St.

1912. Rodman, John Stewart, M.D., Lecturer on Surgery in the Medico-Chirurgical College; Assistant Surgeon to the Medico-Chirurgical Hospital; Assistant Surgeon to the Presbyterian Hospital; Assistant Surgeon to the Outpatient Department of the Pennsylvania Hospital. 1310 Medical Arts Building.

1909. Rosenberger, Randle C., M.D., Professor of Hygiene and Bacteriology in the Jefferson Medical College. 2330 N. Thirteenth St.

‡1898. Ross, George G., M.D., Visiting Surgeon, Germantown, Stetson and Methodist Hospitals; Assistant Surgeon, Lankenau Hospital; Assistant Surgeon, Hospital of the University of Pennsylvania; Associate in Surgery, University of Pennsylvania School of Medicine. 1721 Spruce St.

*1907. Royer, B. Franklin, M.D., American Child Health Association. 370 Seventh Ave., New York City.

†1905. Rugh, James Torrance, A.B., M.D., Clinical Professor of Orthopedic Surgery in the Woman's Medical College of Pennsylvania; Orthopedic Surgeon to the Jefferson Medical College; Orthopedic Surgeon to the Methodist and the Philadelphia General Hospitals. 1616 Spruce St.

ELECTED

1897. Sailer, Joseph, Ph.B., M.D., Professor of Clinical Medicine in University of Pennsylvania; Physician to the Philadelphia General, the University, and Presbyterian Hospitals. 250 S. Eighteenth St.

1900. Sajous, Charles E. de M., M.D., LL.D., ScD., Knight of the Legion of Honor of France; and of the Order of Leopold of Belgium; Professor of Therapeutics in Temple University Medical School. 2043 Walnut St.

1905. Sartain, Paul J., A.M., M.D. 2006 Walnut St.

1908. Sautter, Albert C., M.D., Assistant in the Dispensary for Diseases of the Eye in the University Hospital; Assistant in the Eye Dispensary of the Lankenau Hospital, 1421 Locust St.

1906. Saylor, Edwin S., M.D., Chief Ophthalmologist to the Diagnostic Hospital of Philadelphia. 2100 Chestnut St.

1920. Scarlett, Hunter W., M.D. 230 S. Twenty-first St.

*1910. Scarlett, Rufus B., M.D., Laryngologist to the Home for Consumptives at Chestnut Hill; Assistant in the Department for Diseases of the Ear, Throat, and Nose of the Pennsylvania Hospital; Instructor in Diseases of the Nose and Throat in the Philadelphia Polyclinic and College for Graduates in Medicine. 78 N. Clinton Ave., Trenton, N. J.

1917. Schaeffer, Jacob Parsons, A.M., M.D., Ph.D., Professor of General Anatomy and Director of the Daniel Baugh Institute of Anatomy of the Jefferson Medical College. 4634 Spruce St.

1899. Schamberg, Jay F., M.D., Professor of Dermatology and Syphilology, Graduate School of Medicine, University of Pennsylvania; Director, Research Institute of Cutaneous Medicine. 1402 Spruce St.

1917. Schnabel, Truman Gross, A.B. (Lehigh), M.D., Associate in Medicine, University of Pennsylvania; Assistant Physician, Philadelphia General Hospital; Physician in the Medical Out-patient Department, University Hospital. 1704 Pine Street.

1917. Schoff, Charles H., M.D. Media, Pa.

1921. Schumann, Edward Armin, A.B., M.D., Obstetrician and Gynecologist, Philadelphia General Hospital; Gynecologist, Frankford Hospital; Lecturer on Obstetrics, Jefferson Medical College; Consulting Gynecologist, Rush Hospital. 1814 Spruce St.

ELECTED

1887. de Schweinitz, George Edmund, A.M., M.D., LL.D., Professor of Ophthalmology in the University of Pennsylvania; Consulting Ophthalmic Surgeon to the Orthopædic Hospital and Infirmary for Nervous Diseases, The Philadelphia Polyclinic and School for Graduates in Medicine, and the Philadelphia General Hospital; Ophthalmic Surgeon to the University Hospital. 1705 Walnut St.

*1913. de Schweinitz, George Lord, B.S., M.D., Assistant in the Department of Ophthalmology, St. Luke's Hospital, Bethlehem, Pa. 85 East Broad St., Bethlehem, Pa.

1910. Schwenk, Peter N. K., M.A., M.D., Attending Surgeon to the Eye Department of the Pennsylvania Hospital; Attending Surgeon to the Wills Eye Hospital. 1417 N. Broad Street.

1917. Service, Charles A., M.D. Bala, Pa.

1908. Shannon, Charles E. G., A.B., M.D., Instructor in Ophthalmology in the Jefferson Medical College; Assistant in the Ophthalmological Clinic at the Jefferson Medical College Hospital; Ophthalmologist to the Seybert Institution. N. E. Corner Seventeenth and Walnut Sts.

1897. Sharpless, William T., M.D., Physician to the Chester County Hospital. West Chester, Pa.

1906. Shields, William G., M.D., Dermatologist to Germantown Hospital; Chief of Dermatological Clinic and Assistant Physician to the Jewish Hospital. 414 School Lane, Germantown.

*1908. Shoemaker, Harlan, A.B., M.D., Lecturer in Surgery, University of Southern California; Surgeon to Los Angeles County Hospital; Surgeon to Washington Street Clinic. 521 Bank of Italy Building, Los Angeles, Calif.

†1893. Shoemaker, Harvey, M.D., Visiting Physician to the Sheltering Arms; Consulting Physician to the Southern Home for Destitute Children; Assistant Physician to the Lankenau Hospital; Physician to the Out-patient Department of the Lankenau Hospital. 1727 Spruce St.

†1896. Shoemaker, William T., M.D., Professor of Ophthalmology, Graduate School of Medicine, University of Pennsylvania; Clinical Professor of Ophthalmology in the Woman's Medical College of Pennsylvania; Ophthalmic Surgeon to Lankenau, Germantown and Pennsylvania Hospitals. 109 S. Twentieth St.

ELECTED

1900. Shumway, Edward Adams, B.S., M.D., Ophthalmic Surgeon to the Philadelphia General, Lankenau and Children's Hospitals; Assistant Professor of Ophthalmology in the University of Pennsylvania, and Assistant Ophthalmic Surgeon to the University Hospital. 2046 Chestnut St.

1903. Sinclair, John Falconer, M.D., Epidemilogist, Presbyterian Hospital; Physician to the Philadelphia Orphan Asylum and to the Presbyterian Orphanage; Physician to the Home of the Merciful Saviour for Crippled Children. 4103 Walnut St.

1907. Sinkler, Francis Wharton, A.B., M.D., Physician to the Orthopædic Hospital and Infirmary for Nervous Diseases; Physician to the Episcopal Hospital. 1606 Walnut Street.

1902. Siter, E. Hollingsworth, M.D., Genito-urinary Surgeon to the Philadelphia General Hospital. 512 Medical Arts Building.

1904. Skillern, Penn-Gaskell, M.D. 1633 Locust St.

1920. Skillern, Ross Hall, M.D., Professor of Laryngology, Graduate School of Medicine, University of Pennsylvania; Laryngologist to Medico-Chirurgical Hospital. 1928 Chestnut St.

1904. Smith, Allen J., A.M., M.D., Sc.D. (Penna. Coll.), LL.D. (McGill Univ.), Professor of Pathology and of Comparative Pathology, and Director of Courses in Tropical Medicine in the University of Pennsylvania. Medical Laboratories, University of Pennsylvania.

1905. Smith, S. MacCuen, M.D., Professor of Otology in the Jefferson Medical College; Aurist and Laryngologist to the Germantown Hospital; Aurist to the Jewish Hospital; Consulting Aurist to the Memorial Hospital, Roxborough; and to the Oncologic Hospital. 1429 Spruce St.

1919. Smyth, Henry Field, M.D. Wayne, Pa.

1908. Speese, John, M.D., Assistant Professor Surgical Pathology, University of Pennsylvania; Associate Professor in Surgery, Graduate School of Medicine, University of Pennsylvania; Surgeon to the Children's and Presbyterian Hospitals. 2032 Locust St.

1895. Spellissy, Joseph M., A.M., M.D., Visiting Surgeon to St. Joseph's and to the Methodist Episcopal Hospitals; Assistant Surgeon to the Orthopedic Department of the University Hospital. 317 S. Fifteenth St.

ELECTED

1897. Spiller, William G., M.D., Professor of Neurology in the University of Pennsylvania; Clinical Professor of Nervous Diseases in the Woman's Medical College of Pennsylvania; Neurologist to the Philadelphia General Hospital; Consultant Neurologist to the Pennsylvania Hospital. 4409 Pine St.

1894. Stahl, B. Franklin, B.S., Ph.G., M.D., Visiting Physician to St. Agnes' and the Philadelphia General Hospitals. 1727 Pine St.

1909. Starbuck, J. Clinton, M.D., Physician to the Orphanage, Orphan Society of Philadelphia at Wallingford and to the Glen Mills School, Glen Mills, Pa. 42 E. Washington St., Media, Pa.

*1875. Starr, Louis, M.D., LL.D. (Haverford). Care of Brown, Shipley & Co., London, England.

1912. Stauffer, Nathan Pennypacker, D.D.S., M.D., Laryngologist and Otologist to the Presbyterian Hospital; to the Philadelphia General Hospital, Tubercular Department; to the Home for Crippled Children. 2015 Walnut St.

1910. Stellwagen, Thomas C., Jr., M.D., Chief Clinical Assistant in the Out-patient Surgical Department of the Jefferson Medical College Hospital. 1912 Pine St.

1895. Stengel, Alfred, M.D., Sc.D., Professor of Medicine in the University of Pennsylvania; Physician to the University Hospital and the Pennsylvania Hospital. 1728 Spruce St.

1901. Stevens, Arthur A., M.D., Professor of Materia Medica, Therapeutics, and Clinical Medicine in the Woman's Medical College of Pennsylvania; Lecturer on Physical Diagnosis in the University of Pennsylvania; Physician to the Episcopal and St. Agnes' Hospitals. 314 S. Sixteenth Street.

1914. Stewart, Thomas S., B.S. (Univ. of Penna.), M.D. 301 S. Eighteenth St.

1921. Stoner, William H., A.B., A.M., Ph.C., Pharm. D., M.D., Biochemist, Philadelphia Hospital; Associate Professor of Biochemistry, Graduate School of Medicine, University of Pennsylvania. Medical Laboratories, University of Pennsylvania.

1898. Stout, George C., M.D., Consulting Laryngologist and Aurist to the Presbyterian Hospital, the Children's Aid Society, and the William Penn Charter School. 2015 Walnut St.

ELECTED

1923. STRECKER, EDWARD, A., A.M., M.D., Medical Director of Department for Mental and Nervous Diseases and Neurologist to Pennsylvania Hospital and Chief of the Out-patient Neuro-psychiatric Clinic; Neurologist, Misericordia Hospital; Assistant Professor of Psychiatry, Graduate School of Medicine, University of Pennsylvania. 4401 Market St.

1923. STROUD, WILLIAM D., M.D. 1011 Clinton St.

1923. STROUSE, FREDERICK M., M.D. 1301 Spruce St.

1884. STRYKER, SAMUEL S., M.D., Consulting Physician to the Presbyterian Hospital. 3833 Walnut St.

*1900. SWAN, JOHN M., M.D., Attending Physician, Park Avenue Clinical Hospital. 457 Park Avenue, Rochester, New York.

1898. SWEET, WILLIAM M., M.D., Clinical Professor of Ophthalmology in the Jefferson Medical College, and Ophthalmic Surgeon to the Jefferson Medical College Hospital; Consulting Surgeon to the Wills Eye Hospital. 1205 Spruce Street.

1900. TALLEY, JAMES ELY, A.B., M.D., Professor of Cardiology, Graduate School of Medicine, University of Pennsylvania; Physician to the Presbyterian and Methodist Episcopal Hospitals. 1811 Spruce St.

1886. TAYLOR, JOHN MADISON, A.B. and A.M. (Princeton), M.D., Professor of Physical Therapeutics and Diatetics, Medical Department Temple University. 1504 Pine St.

1887. TAYLOR, WILLIAM J., M.D., Surgeon to the Orthopædic Hospital and Infirmary for Nervous Diseases, and to St. Agnes' Hospital; Consulting Surgeon to the West Philadelphia Hospital for Women. 1825 Pine St.

1886. TAYLOR, WILLIAM L., M.D. 2223 Tioga St.

†1910. THOMAS, BENJAMIN A., A.B., A.M., M.D., Professor of Genito-urinary Surgery in the Philadelphia Polyclinic and College for Graduates in Medicine; Genito-urinary Surgeon, Presbyterian Hospital; Instructor in Surgery in the University of Pennsylvania; Surgeon-in-Chief, Genito-urinary Department of the Out-patient Department of the University Hospital. 116 S. Nineteenth St.

1912. THOMAS, FRANK WISTER, A.M., M.D., Visiting Physician to "Buttercup Cottage;" Consulting Physician to the Germantown Hospital. 27 E. Mt. Airy Ave.

ELECTED

1907. Thomas, Thomas Turner, M.D., Associate Professor of Applied Anatomy, and Associate in Surgery in the University of Pennsylvania; Surgeon to the Philadelphia General Hospital; Assistant Surgeon to the University Hospital. 2009 Spruce St.

1896. Thorington, James, A.M., M.D., Ophthalmic Surgeon to the Presbyterian Hospital; Ophthalmologist to the Ellwyn, Pa., Training School for Feeble-minded Children. 2031 Chestnut St.

1898. Thornton, Edward Q., M.D., Assistant Professor of Materia Medica in the Jefferson Medical College. 1331 Pine St.

1912. Torrey, Robert G., M.D., Assistant Physician to the Philadelphia General Hospital; Physician to the State Tuberculosis Dispensary. 1716 Locust St.

1896. Toulmin, Harry, M.D., Vice-President, Medical Department, Penn Mutual Life Insurance Company. Haverford, Pa.

1908. Tracy, Stephen E., M.D., Gynecologist to the Stetson Hospital; Visiting Surgeon to the Gynecean Hospital. 1527 Spruce St.

*1901. Tucker, Henry, M.D. Bellevue, Md.

†1894. Tunis, Joseph Price, M.D., Assistant Professor of Histology and Embryology, Temple University. Chestnut Hill, Philadelphia.

1923. Turner, Creighton H., M.D., Associate in Medicine, Jefferson Medical College; Visiting Physician, St. Agnes' Hospital. Medical Arts Building.

1901. Turner, John B., M.D., Ph.D., Assistant in the Ophthalmological Clinic, Methodist Episcopal Hospital. 1833 Chestnut St.

1897. Tyson, T. Mellor, M.D., Physician to the Philadelphia General Hospital; Physician to the Rush Hospital, the Philadelphia Lying-in-Charity Hospital, and the Children's Aid Society of Philadelphia. 1506 Spruce St.

1907. Ullom, Josephus Tucker, M.A., M.D., Physician to the Chestnut Hill Hospital. Carpenter and Quincy Streets, Germantown.

*1913. Vail, William Penn, B.S., M.S., M.D., Laryngologist to the Pennsylvania Institution for the Instruction of the Blind; Laryngologist to the Department for Tuberculosis

ELECTED

of the Philadelphia General Hospital; Assistant Laryngologist and Otologist to the Out-patient Department of the Pennsylvania Hospital; Assistant Laryngologist to the Children's Hospital. Blairstown, N. J.

°1873. Van Harlingen, Arthur, Ph.B. (Yale), M.D., Emeritus Professor of Diseases of the Skin in the Philadelphia Polyclinic. 1831 Chestnut St.

1903. Van Pelt, William Turner, M.D. Chestnut Hill, Philadelphia.

1893. Vansant, Eugene Larue, M.D. 2046 Chestnut St.

1912. Vaux, Norris Wistar, M.D., Surgeon to the Chestnut Hill Hospital; Surgeon to the Dispensary of the Germantown Hospital. 1810 S. Rittenhouse Square.

*1897. Veasey, Clarence A., A.M., M.D. Suite 404, Paulsen Building, Spokane, Wash.

†1883. Vinton, Charles Harrod, A.M., M.D. P. O. Box 464, Atlantic City, N. J.

1903. Wadsworth, William Scott, M.D. 3914 Baltimore Ave.

1906. Walker, John K., M.D., Physician to the Children's Hospital of the Mary J. Drexel Home; Physician to the Out-patient Department of the Pennsylvania Hospital. 1819 De Lancey St.

1907. Walker, Warren, M.D., Dermatologist to the Episcopal and Bryn Mawr Hospitals. 1819 Chestnut St.

1904. Walsh, Joseph, A.M., M.D., Visiting Physician to and Medical Director of the White Haven Sanatorium; Medical Director of St. Agnes' Hospital. Medical Arts Building.

1910. Ward, E. Tillson, A.M., M.D. 2006 Mt. Vernon St.

1895. Watson, Arthur W., M.D., Professor of Diseases of the Throat and Nose in the Philadelphia Polyclinic and College for Graduates in Medicine; Laryngologist to the Jewish Hospital; Laryngologist and Aurist to the Mt. Sinai Hospital; Laryngologist to Home for Incurables. 2013 Locust St.

1886. Watson, Edward W., M.D., Physician to the Magdalene Home. 38 S. Nineteenth St.

1923. Watson, William R., M.D., Chief Laryngologist, Episcopal and Diagnostic Hospitals; Assistant Laryngologist, Pennsylvania Hospital. 2124 Pine St.

1903. Weber, Charles H., M.D., Associate in Pediatrics, Graduate School of Medicine, University of Pennsylvania; Physician to the Medical Dispensary, Children's Hospital; Pediatrist,

ELECTED

Howard Hospital and to the Municipal Court. 1811 Spruce Street.

1920. Weidman, Fred. D., M.D., Assistant Director, Laboratory of Dermatological Research, University of Pennsylvania; Assistant Pathologist, Philadelphia Zoölogical Garden and University Hospital. 20 Tenby Road, Llanerch, Pa.

1906. Weisenburg, Theodore H., M.D., Professor of Neurology, Graduate School of Medicine, University of Pennsylvania; Neurologist, Philadelphia General Hospital and Orthopædic Hospital and Infirmary for Nervous Diseases. 1909 Chestnut St.

1923. Weiss, Edward, M.D., Demonstrator of Clinical Medicine, Jefferson Medical College; Assistant Attending Physician, Jewish Hospital and in the Tuberculosis Wards, Philadelphia General Hospital. 1906 Walnut St.

1913. Wells, P. Frailey, A.M., M.D., Ph.D., 754 N. Fortieth St.

1893. Westcott, Thompson S., M.D., Associate in Diseases of Children in the University of Pennsylvania; Pediatrist to the Methodist Episcopal Hospital; Consulting Physician, Haddock Memorial Home. 1720 Pine St.

1884. Wharton, Henry R., M.D., Consulting Surgeon to the Presbyterian and the Children's Hospitals; Surgeon to Girard College; Consulting Surgeon to the Bryn Mawr Hospital, the Chestnut Hill Hospital, St. Christopher's Hospital for Children, the Pennsylvania Institution for the Deaf and Dumb, and the Pennsylvania Institution for the Blind. Berwyn, Pa.

1921. Whitaker, William, M.D., Surgeon to Dispensary of the Episcopal Hospital. 5448 Germantown Ave.

1901. White, Courtland Y., M.D., Director of the Pathological Laboratories of the Episcopal Hospital; Pathologist to the Children's Hospital and to the Kensington Hospital for Women; Chief Bacteriologist to the Bureau of Health, Department of Health and Charities, Philadelphia. 6611 N. Tenth St.

*1905. Whiteway, Harold M., M.D. 2356 W. Twenty-first St., Los Angeles, Calif.

1898. Whiting, Albert D., M.D., Associate Professor of Surgery, Graduate School of Medicine, University of Pennsylvania; Associate Surgeon, Lankenau Hospital; Surgeon, Germantown Hospital. 1523 Spruce St.

ELECTED

1914. Willard, de Forest P., B.S. (Univ. of Penna.), M.D., Professor of Orthopædics, Graduate School of Medicine, University of Pennsylvania; Orthopædic Surgeon to the Polyclinic, Jewish and Delaware Hospitals. 1630 Spruce St.

1907. Williams, Carl, B.S., M.D., Ophthalmic Surgeon to the Germantown Hospital; Instructor in Ophthalmology in the University of Pennsylvania; Ophthalmic Surgeon to the Pennsylvania Institution for the Deaf and Dumb. School Lane and Greene Sts., Germantown.

1920. Williams, Philip F., Ph.B., M.D., Assistant Professor of Obstetrics, Graduate School of Medicine, University of Pennsylvania; Instructor in Obstetrics, School of Medicine, University of Pennsylvania; Obstetrician to the Maternity Hospital of Philadelphia and Assistant Gynecologist to the Presbyterian Hospital. 2206 Locust St.

1916. Wilmer, Harry B., M.D., Assistant Instructor in Medicine, University of Pennsylvania; Assistant Visiting Physician and Assistant Neurologist to the Germantown Hospital; Visiting Chief to the Dispensary of the Germantown Hospital. 138 W. Walnut Lane, Germantown.

1921. Wilson, George, M.D., Instructor in Medicine and Neurology, School of Medicine, University of Pennsylvania; Assistant Physician, Philadelphia Hospital; Assistant Neurologist, Philadelphia, University and Children's Hospitals; Consulting Neurologist, St. Christopher's Hospital and the Pennsylvania Training School for Feeble-minded Children. 1909 Chestnut St.

1874. Wilson, James Cornelius, A.M. (Princeton), M.D., Emeritus Professor of the Practice of Medicine and of Clinical Medicine in the Jefferson Medical College; Physician-in-Chief to the Lankenau Hospital; Emeritus Physician to the Pennsylvania Hospital; Consulting Physician to the Bryn Mawr Hospital. 1509 Walnut St.

1902. Wilson, Samuel M., M.D. 2018 Chestnut St.

1897. Wilson, W. Reynolds, M.D. 1709 Spruce St.

1904. Wister, James W., M.D., Physician to the Out-patient Department of the Germantown Hospital. 5430 Germantown Ave.

*1901. Witmer, A. Ferree, M.D. Freeport, Long Island, N. Y.

1918. Wolferth, Charles Christian, A.B., M.D., Associate in Medicine, University of Pennsylvania; Associate in Cardiac

ELECTED

Pathology, William Pepper Laboratory of Clinical Medicine, University of Pennsylvania. 1704 Pine St.

1893. Wood, Alfred C., M.D., Assistant Professor of Surgery in the University of Pennsylvania; Surgeon to the University, the Philadelphia General, St. Timothy's, and the Howard Hospitals. 2035 Walnut St.

1900. Wood, George, B., M.D., Laryngologist and Otologist, Orthopædic and Howard Hospitals; Professor of Laryngology, Graduate School, University of Pennsylvania. 1830 Spruce St.

1903. Wood, Horatio C., Jr., M.D., Professor of Pharmacology and Therapeutics in the University of Pennsylvania; Professor of Materia Medica, Philadelphia College of Pharmacy and Science. 2009 Chestnut St.

1880. Woodbury, Frank, M.D., Secretary to the Committee on Lunacy of the Board of Charities of Pennsylvania. 3345 N. Seventeenth St.

*1911. Woods, Andrew H., A.B., M.D., Professor and Head of the Neurological Department, Peking Union Medical College, Peking, China.

†1897. Woodward, George, M.D. W. Willow Grove Ave., Chestnut Hill, Philadelphia.

1913. Woodward, W. Wellington, M.D. 26 S. Church St., West Chester, Pa.

1903. Worden, Charles B., A.M., M.D. Princeton, N. J.

‡1889. Young, James K., M.D., Professor of Orthopædics, Graduate School of Medicine, University of Pennsylvania; Orthopædic Surgeon, Philadelphia General Hospital; Consulting Orthopædic Surgeon, Woman's Hospital and Philadelphia Lying-in-Charity. 222 S. Sixteenth St.

1894. Zentmayer, William, M.D., Professor of Ophthalmology, Graduate School of Medicine, University of Pennsylvania; Attending Surgeon to the Wills Eye Hospital; Ophthalmologist to the Glen Mills School. 1506 Spruce St.

1899. Ziegler, S. Lewis, A.M., M.D., LL.D., Attending Surgeon to the Wills Eye Hospital; Chief Ophthalmic Surgeon to St. Joseph's Hospital; Membre Société Française d'Ophthalmologie. 1625 Walnut St.

1887. Ziegler, Walter M. L., A.M., M.D. 1418 N. Seventeenth St.

1895. Zimmerman, Mason W., M.D., Consulting Ophthalmic Surgeon to the Germantown Hospital. 1518 Waverly St.

ASSOCIATE FELLOWS

(Limited to Fifty, of whom Twenty may be Foreigners)

American

ELECTED

1911. Abbe, Robert, M.D. 40 W. Fifty-ninth St., New York City, New York.

1909. Billings, Frank, M.D., 1550 North State Parkway, Chicago, Illinois.

1893. Councilman, William T., M.D., Harvard Medical College, Boston, Massachusetts.

1909. Crile, George W., M.D., 1021 Prospect Avenue, S. E., Cleveland, Ohio.

1909. Dana, Charles Loomis, M.D., 53 West Fifty-third Street, New York City, New York.

1892. Emmet, Thomas Addis, M.D., 91 Madison Avenue, New York City, New York.

1912. McCaw, Brig.-Gen. Walter D., M.D. Assistant Surgeon-General, U. S. A., Army Medical School, Washington, D. C.

1906. Mayo, William J., M.D., Rochester, Minnesota.

1906. Pilcher, Lewis Stephen, M.D., 145 Gates Avenue, Brooklyn, New York.

1906. Shattuck, Frederick C., M.D., 135 Marlborough Street, Boston, Massachusetts.

1894. Warren, J. Collins, M.D., 58 Beacon Street, Boston, Massachusetts.

1894. Weir, Robert F., M.D., 1155 Park Avenue (Ninety-second Street), care of Mrs. Ed. La Montayne, New York City, New York.

1892. Welch, William H., M.D., Johns Hopkins Hospital, Baltimore, Maryland.

Foreign

1890. Baccelli, Guido, Rome, Italy.
1908. Bannerman, W. B., M.D., General, I. M. S., 11 Strathearn Place, Edinburgh, Scotland.
1909. Macallum, Archibald B., M.A., M.B., Ph.D., Sc.D., LL.D., F.R.S., Department of Biochemistry, McGill University, Montreal, Canada.
1922. McCarrison, Lieut.-Col., Robert, M.D. (London), D.Sc. (Oxford, Eng.), LL.D., C. I. E., The Neuk, Silverdale Road, Eastbourne, England.
1906. Myles, Sir Thomas, M.D., 33 Merion Square, W., Dublin, Ireland.
‡1898. Roddick, Thomas G., M.D., 80 Union Avenue, Montreal, Canada.
1908. Ross, Col. Sir Ronald, K.C.B., K.C.M.G., F.R.S., M.D., D.Sc., LL.D., 36 Harley House, London, N. W. I., England.

‡ Deceased.

CORRESPONDING MEMBERS

1880. Carrow, Flemming, M.D., 823 David Whitney Building, Detroit, Mich.
1885. Rendu, Jean, M.D., Lyons, France.
1916. Estes, William Lawrence, M.D., South Bethlehem, Pa.
1916. Guitéras, John, M.D., Benavides, Cuba.
1917. Miner, Charles H., M.D., Wilkes-Barre, Pa.
‡1917. Stevens, Cyrus L., M.D., Athens, Pa.

‡ Deceased.

NECROLOGICAL LIST

FELLOWS

GEORGE G. ROSS, M.D.	December 27, 1922
WILLIAM H. MORRISON, M.D.	December 31, 1922
GUTHRIE McCONNELL, M.D.	January 5, 1923
JAMES DARRACH, M.D.	January 17, 1923
CHARLES LINCOLN FURBUSH, M.D.	June 26, 1923
JAMES KELLY YOUNG, M.D.	August 28, 1923
JAMES CHESTON MORRIS, M.D.	November 29, 1923
HENRY LEAMAN, M.D.	December 20, 1923

ASSOCIATE FELLOW

SIR THOMAS G. RODDICK, M.D.	March 20, 1923

CORRESPONDING MEMBER

CYRUS L. STEVENS, M.D.	February 19, 1923

LIST OF PRIZES AND LECTURES

THE WILLIAM F. JENKS MEMORIAL PRIZE

(Triennial)

1889	John Strahan, M.D.	Belfast (Ireland).
1895	Abram Brothers, M.D.	New York.

NOTE—June 12, 1900, the William F. Jenks Memorial Prize Fund was transferred to the Library as the "William F. Jenks Memorial Library Fund."

ALVARENGA PRIZE OF THE COLLEGE OF PHYSICIANS OF PHILADELPHIA

(Annual)

1890	R. W. Philip, M.D.	Edinburgh (Scotland).
1891	L. Duncan Bulkley, M.D.	New York.
1892	R. H. L. Bibb, M.D.	Saltillo.
1894	G. E. de Schweinitz, M.D.	Philadelphia.
1895	Guy Hinsdale, M.D.	Philadelphia.
1897	Joseph Collins, M.D.	New York.
1898	S. A. Knopf, M.D.	New York.
1898	Robert Randolph, M.D.	Baltimore.
1900	David de Beck, M.D.	Cincinnati.
1901	George W. Crile, M.D.	Cleveland.
1903	William S. Carter, M.D.	Galveston.
1905	D. Chalmers Watson, M.D.	Edinburgh (Scotland).
1907	William Louis Chapman, M.D.	Providence.
1908	William T. Shoemaker, M.D.	Philadelphia.
1910	M. Katzenstein, M.D.	Berlin (Germany).
1911	Francis D. Patterson, M.D.	Philadelphia.
1914	H. B. Sheffield, M.D.	New York.
1915	J. E. Sweet, M.D.	Philadelphia.
1917	Wilburt C. Davison, M.D.	Baltimore.
1921	John W. Churchman, M.D.	New York
1923	Edward P. Heller, M.D.	Kansas City, Mo.

NATHAN LEWIS HATFIELD PRIZE FOR ORIGINAL RESEARCH IN MEDICINE

(Triennial)

1901	Henry F. Harris, M.D.	Atlanta.
1909	Martin Henry Fischer, M.D.	Oakland.
1917	A. B. Macallum, M.D., F.R.S.	Toronto.
1919	Harvey Cushing, M.D.	Boston.
1921	William R. Nicholson, M.D.	Philadelphia.
1922	Sir Thomas Lewis, C.B.E., M.D., F.R.S.	London (Eng.).
1923	Frederick Grant Banting, M.D.,	Toronto.

NOTE.—November 29, 1913, by Supplemental Deed of Trust, the title of this Fund was changed to "Nathan Lewis Hatfield Prize and Lectureship."

WEIR MITCHELL LECTURES

Jan. 17, 1911	Arthur R. Cushny, M.D.	London.
Mar. 30, 1911	Edmund B. Wilson, Ph.D., LL.D.	New York.
May 16, 1911	Svante Arrhenius	Stockholm.
Nov. 3, 1911	William T. Porter, M.D.	Boston.
Mar. 29, 1912	William H. Howell, M.D.	Baltimore.
Oct. 21, 1912	G. H. F. Nuttall, F.R.S., M.D.	Cambridge (Eng.).
April 4, 1913	H. P. Armsby, Ph.D., LL.D.	Pennsylvania.
Feb. 25, 1914	Harvey Cushing, M.D.	Boston.

PUBLIC LECTURES

Feb. 16, 1910	S. Weir Mitchell, M.D.	Philadelphia.
Nov. 17, 1910	Simon Flexner, M.D.	New York.
Dec. 15, 1910	William H. Welch, M.D.	Baltimore.
April 18, 1911	James G. Mumford, M.D.	Clifton Springs, N.Y.
Nov. 20, 1911	Talcott Williams, A.M., LL.D., Litt.D.	Philadelphia.
April 29, 1912	Owen Wister, A.M., LL.D.	Philadelphia.
Feb. 17, 1913	John K. Mitchell, M.D.	Philadelphia.
Feb. 15, 1916	Daniel J. McCarthy, M.D. and Walter Estell Lee, M.D.	Philadelphia.
April 15, 1916	Surgeon A. M. Fauntleroy	U. S. Navy.

MARY SCOTT NEWBOLD LECTURES

Mar. 29, 1917	A. T. McCormack, M.D.	Bowling Green, Ky.
April 4, 1919	Col. Thomas W. Salmon, M.C.,	U. S. Army.
Feb. 6, 1920	Alonzo E. Taylor, M.D.	Philadelphia.
Feb. 4, 1921	Maj.-Gen. Merritte W. Ireland	Washington.
April 20, 1921	Prof. William Romaine Newbold,	Philadelphia.
Nov. 11, 1921	Robert McCarrison, M.D., D.Sc., LL.D.	Oxford (Eng.).
Feb. 1, 1922	Hans Zinsser, M.D.	New York.
April 5, 1922	G. Canby Robinson, M.D.	Baltimore.
Dec. 6, 1922	Donald C. Balfour, M.D.	Rochester, Minn.
Mar. 7, 1923	J. J. R. Macleod, M.D.	Toronto.
April 4, 1923	Prof. Leon Asher, M.D.	Berne (Switzerl'd).

MÜTTER LECTURES

1865	J. H. Packard, M.D.	Philadelphia.
1866	J. H. Packard, M.D.	Philadelphia.
1867	J. H. Packard, M.D.	Philadelphia.
1868	Harrison Allen, M.D.	Philadelphia.
1869	J. H. Brinton, M.D.	Philadelphia.
1872	J. da S. Solis Cohen, M.D.	Philadelphia.
1879	S. W. Gross, M.D.	Philadelphia.
1882	E. O. Shakespeare, M.D.	Philadelphia.
1885	H. F. Formad, M.D.	Philadelphia.
1888	O. H. Allis, M.D.	Philadelphia.
1890–1891	Roswell Park, M.D.	Buffalo.
1893–1894	De Forrest Willard, M.D., and Guy Hinsdale, M.D.	Philadelphia.
1896	O. H. Allis, M.D.	Philadelphia.
1899–1900	J. B. Roberts, M.D.	Philadelphia.
1901	H. W. Cushing, M. D.	Boston.
1902	L. A. La Garde, M.D.	Washington.
1903	C. N. B. Camac, M.D.	New York.
1904	G. H. Monks, M.D.	Boston.
1905	A. O. J. Kelly, M.D.	Philadelphia.
1906	W. J. Mayo, M.D.	Rochester, Minn.
1907	J. Rogers, M.D., and S. P. Beebe, M.D.	New York.
1908	G. W. Crile, M.D.	Cleveland.
1909	H. D. Fry, M.D,	Washington.
1910	T. W. Hastings, M.D.	New York.

1911	C. F. Nassau, M.D.	Philadelphia.
1912	J. C. Bloodgood, M.D.	Baltimore.
1913	R. C. Coffey, M.D.	Portland, Ore.
1914	F. H. Albee, M.D.	New York.
1915–1916	Rudolph Matas, M.D.	New Orleans, La.
1916	Nelson M. Percy, M.D.	Chicago, Ill.
1917	Chevalier Jackson, M.D.	Philadelphia.
1919	Alexis V. Moschcowitz, M.D.	New York.
1920	J. Chalmers Da Costa, M.D.	Philadelphia.
1921	Professor H. C. Jacobaeus	Stockholm (Swed.).
1922	James Ewing, M.D.	New York.
1923	J. Edwin Sweet, M.D.	Philadelphia.

WEIR MITCHELL ORATION

1919	Charles W. Burr, M.D.	Philadelphia.
1922	Charles Loomis Dana, M.D	New York.

MEMOIR OF JAMES W. HOLLAND, M.D.[1]

By ALBERT P. BRUBAKER, M.D.

It was deemed wise and proper by our predecessors in the College of Physicians that on the occasion of the death of a Fellow the President should appoint an associate to prepare a memoir of the deceased which should present a brief account of his life, his achievements and his personal characteristics. The President, following this long-established custom, has honored me with the request to prepare such a memoir of our late Fellow, Prof. James W. Holland, who died February 10, 1922.

Though not an active participant in the activities of the College by reason of his special calling, he was frequently present at the meetings and an appreciative visitor to the library. His kindly manner and uniform courtesy endeared him to those with whom he was brought into personal relations.

Dr. James W. Holland was born April 24, 1849, in Nashville, Tenn. He was the son of Dr. Robert Chappel Holland and Elizabeth Lewis (Turner) Holland. Owing to an epidemic of cholera which made its appearance in 1852, the family moved to Louisville, Ky. In the schools of that city his early education was received, after which he entered the University of Louisville, and in 1865 was graduated a Bachelor of Arts. By reason of his attainments and subsequent work, he received the degree of A.M. three years later. During this latter period he attended two courses of lectures in the medical department of the University of Louisville.

[1] Read December 5, 1923.

In the fall of 1867 he entered the Jefferson Medical College of Philadelphia, from which he was graduated in the spring of 1868, receiving the degree of Doctor of Medicine.

After his graduation, Dr. Holland returned to his home in Louisville and began, in association with his father, the practice of medicine, in which he continued for a series of years, with eminent success. Possessing the instinct and capabilities of the teacher, he soon connected himself with the teaching staff of the University and received an appointment as assistant demonstrator of anatomy.

Prof. Holland's acquaintance with the facts of chemical science and with the intimate relations of chemistry to the problems of practical medicine, combined with his aptitude for teaching, led, in 1872, to his election to the chair of medical chemistry in the University of Louisville, a position he occupied with success and distinction through a series of years. Subsequently, as other chairs became vacant, he successively occupied the chair of materia medica, and eventually that of the practice of medicine.

During his residence in Louisville, Prof. Holland was a member of the Louisville College of Physicians and Surgeons, of the Louisville Medical and Chirurgical Society, President of the Kentucky State Medical Society and a member of the American Medical Association. In 1878 he was appointed a member of the Kentucky State Board of Health. Though deeply occupied with the duties of his various positions, Prof. Holland found time to contribute papers of a medical character to the current medical journals.

In 1877 Dr. Holland married Mary B., daughter of E. W. Rupert, Esq., of Louisville, Ky.

The inherent and growing interest of Prof. Holland not only in the science of chemistry, but to a far greater degree in its relations to the problems of clinical medicine, won for him a wide and enviable reputation as a successful exponent and teacher of medical chemistry in the University of Louisville. For these reasons, when the chair of medical chemistry and toxicology became vacant through the retirement of Prof. Robert E. Rogers, in 1885, the Board of Trustees of Jefferson Medical College elected Prof. Holland to the chair, a position that he worthily occupied for a

period of twenty-seven years. After his resignation he was elected emeritus professor.

In 1887 Prof. Holland was called on to assume, in addition to his professional work, the duties of the office of Dean, in which capacity he served the College for a period of twenty-five years, when he was relieved of many of the exacting duties by the subdean, Dr. Ross V. Patterson. A few years later, in 1916, Prof. Holland finally resigned his position as Dean. In recognition of his services to science and the College the Board of Trustees conferred upon him the honorary degree of Doctor of Science.

Though retiring from active duties in the College, Prof. Holland maintained his interest in all that pertained to its welfare and generously gave of his time and advice whenever called on to do so.

Prof. Holland as a teacher and lecturer was eminently successful. Possessing a thorough and accurate knowledge of his subject, endowed with a clear and well-modulated voice and a distinct enunciation, and gifted with the art of presenting the essential facts in a clear and forceful manner, his lectures were always attractive and instructive. As his lectures were intended, as he felt they should be in a medical school, to apply the results of chemical research to the solution of the problems of clinical medicine, he elicited the interest and attention of the student body without effort. The didactic lectures were supplemented and illustrated by ingeniously devised experiments, which were novel and striking and generally successful by reason of the care with which they were always prepared.

As Dean of the Faculty for twenty-nine years, Prof. Holland performed his duties with sincerity and fidelity, and always with reference to the welfare of the College and the student body. In the conduct of this office he displayed executive ability of a high order and contributed in large measure in determining the policy of the College from year to year and in accordance with changing methods of medical education. His keen perceptions enabled him to adapt that policy, as occasion demanded, to the various legislative enactments relating to undergraduate instruction and medical practice. During the period of his incumbency of the

Dean's office the Jefferson Medical College grew and advanced from year to year and established itself as one of the leading medical institutions of the country, devoting itself to undergraduate instruction. The success that it has attained is in large part due to his wisdom and his untiring energy in promoting its highest interests.

Kind in manner, gracious in personality, loyal in friendship, ready in coöperation, tolerant of the views of others, he will ever live in the memory of those with whom he was associated.

Prof. Holland was a member of the College of Physicians, of the American Philosophical Society (and for years one of its secretaries), a member of the Council of Medicine of the American Medical Association from 1907 to 1916 and President of the Association of American Medical Colleges for one term.

Among Prof. Holland's contributions to medical science may be mentioned *Diet for the Sick, Common Poisons and the Urine,* and *Medical Chemistry and Toxicology.*

ANNUAL ADDRESS OF THE PRESIDENT[1]

By THOMAS R. NEILSON, M.D.

Pursuant to custom and in obedience to the By-laws, it becomes the duty of your presiding officer to present at this meeting a review of the work of the College in the past year. At the last meeting you heard the Annual Reports of certain of the officers, the Standing Committees and the Sections. For this reason it is not my purpose to dwell at any length upon the details recounted in these reports, but rather to touch only upon the high spots in the story of the various activities of the College, with perhaps occasional comment where this seems proper.

Of the component parts of the College, that which, in a survey of its activities, naturally at the outset arrests our attention is our wonderful library. The elaborate report of the committee charged with the responsibility of directing the affairs of this department, which will appear in full in the *Transactions*, gives at great length an account of all that has transpired in the year, and reflects the splendid condition, the great number of accessions to and the continued extensive use made of this famous collection of medical books and journals.

Under the vigilance of the Hall Committee, the good condition of our building has been maintained, repairs made to the plaster work in the upper corridor and some needed repainting done. The surrounding grounds were kept in excellent order during the open season, and the attractive plantings of shrubbery and flowers in the splendid garden beside the building added greatly to its handsome and dignified appearance and constituted a beauty spot to attract the attention and please the eye of all passers-by.

[1] Read January 3, 1923.

The Directory for Nurses had a very satisfactory year. The report of its committee tells us that 5137 calls were filled, or more than 14 per day. The number of women nurses registered is 673, while of certificate nurses there are 15. Is it not, perhaps, to be regretted that there are not more of the latter class available? There is a wide field of usefulness for them, for often the services of the so-called certificate nurse would probably answer all the needs of many cases of illness of the less serious type fully as well as the registered nurse, whose highly refined education in her profession is more than equal to the demands of the occassion and whose compensation has now reached a figure almost beyond the resources of the person of average moderate means.

Concerning Finances. The finances of the College, under the capable administration of the Treasurer and the Committee on Finance, continue to be in sound and gratifying condition. By judicious allotment the committee succeeds each year in apportioning the income available for general expenses so as to meet as nearly as possible the requirements of the several departments of the College. The sum available for these general purposes is barely sufficient and some curtailment of the appropriations asked for is usually necessary. With ever-increasing demands, and mounting costs, this condition will continue, unless, through good fortune, relief comes in the form of endowments, the income from which may be devoted to general maintenance alone.

As to the Scientific Business. At the nine meetings held during the year eleven papers were read, eight by Fellows and three by invited guests. In addition, two memoirs of deceased Fellows were read. At the April meeting, Dr. David Riesman read an admirable memoir of Dr. M. Howard Fussell, and at the October meeting Dr. J. Hendrie Lloyd read his excellent memoir of Dr. Charles W. Dulles.

At two of the stated meetings, those of February and April, a Mary Scott Newbold Lecture constituted the scientific program. At the former, Dr. Hans Zinsser, professor of bacteriology and immunology in Columbia University, was the lecturer, his subject

being, "Changes in our Conceptions of Antibody Reactions," and at the April meeting, Dr. G. Canby Robinson, acting professor of medicine in Johns Hopkins University and Dean of the Vanderbilt Medical School, addressed the College on "The Therapeutic Use of Digitalis and its Relation to Experimental Pharmacology." At the November meeting the Mütter Lecture for the year was delivered by Dr. James Ewing, professer of pathology in Cornell University Medical College. His subject was, "The Principles of the Radiation Treatment of Cancer."

Besides the foregoing, there was delivered on May 5, 1922, by Sir Thomas Lewis, of London, the Fourth Nathan Lewis Hatfield Lecture on "The Action of Digitalis on Auricular Fibrillation and Flutter," and on November 23, a "Weir Mitchell Oration" was delivered by Dr. Charles Loomis Dana, of New York, on "Medicine and the Humanities."

Such is the record of the year's scientific business at meetings of the College. Is it not a matter of more than passing import that during the year there were presented only eight papers by Fellows of the College? Why are there not more?

It is true that in the Sections a goodly number of papers are read, and that these may properly be regarded as constituting part of the sum total of the scientific work of the College. But why should not some of these papers be presented at the sessions of the College itself? So strongly convinced was the Council that "the activities of the various Sections are impairing the scientific interest in the College meeting," that at a meeting held on September 26 last it passed a resolution instructing the Committee on Scientific Business to confer with the Chairmen and Clerks of the Sections, with the object in view of obtaining papers of general interest to the meetings of the College. The conference was held and it is hoped that good will result from it.

Surely there must be some papers presented to the different sections which are not specialized to such a degree as to be incomprehensible or devoid of interest at a general session of the College. Indeed, might not some of the papers upon specialized subjects meet with profitable discussion by the general practitioner or the general surgeon?

The Sections of the College. The reports submitted by the several Sections show that three of them—that on General Medicine, that on Ophthalmology and that on Otology and Laryngology—have transacted a very creditable amount of scientific business during the year. The Section on General Medicine reports a membership of 111, a gain of 41 in two years; the Section on Ophthalmology has 39 members; and that on Otology and Laryngology, 28.

The Section on Medical History, with its limited scope, had two very interesting meetings during the year.

The Section on Industrial Medicine and Public Health does not seem to meet with very great success. It reports a membership of only 13—two below the number required by the regulations—and this in spite of earnest effort made by its officers, personally, as well as by a circular letter to the Fellows of the College, soliciting interest in the Section. The Clerk of the Section tells me, however, that he has reason to believe that, in consequence of renewed efforts, there will shortly be an encouraging response and a material increase in membership. It occurs to your President that the question may properly be raised as to whether there is need for both this Section and the Standing Committee on Public Health and Preventive Medicine. Do not their fields to some extent overlap, and might it not be well to dispense with one of them?

Of the Council. In the meetings of the Council, at which the attendance of members is most creditable, all matters referred from the College and other business originating in the Council have received the fullest and most painstaking consideration. Not the least among the duties imposed upon the Council is the action upon Propositions for the honor of Fellowship. Often perplexed for want of the kind of information it should have for intelligent action, and feeling "that the Fellows should exercise great care in proposing physicians for election to Fellowship," the Council adopted, in October last, a resolution stating that:

"In order that he may be recommended by the Council for election to Fellowship in the College, the eligibility for Fellowship of any candidate must be based on evidence presented by his

proposers that he has shown distinct ability in his profession, either by his writings or by other proofs capable of demonstration. Should no such proofs be forthcoming, the Council may, at its discretion, demand that he submit a Thesis for its consideration."

"It shall be the duty of the proposers of any candidate to see that he is known to at least four members of the Council."

In the form of a letter, a copy of this resolution was sent in November last to every Fellow of the College. It is hoped that good will come of this.

Of Membership. The roll of the College on December 1, 1921, included:

482 Fellows; 19 Associate Fellows (13 American and 6 Foreign); 6 Corresponding Members. Total, 507

During the past year 6 Fellows were lost by death:

Dr. J. Morton Boice, elected a Fellow on November 2, 1910, died on December 2, 1921, in the forty-sixth year of his age. He was physician to the Home of the Merciful Saviour for Crippled Children, and for some years served also as secretary of the Philadelphia County Medical Society.

Dr. George Erety Shoemaker, elected a Fellow on November 5, 1890, died on January 5, 1922, in his sixty-fifth year. Early in his professional career, Dr. Shoemaker's inclinations were toward general surgery, and he held appointments as surgeon to the Out-patient Department of the University Hospital and of the Presbyterian Hospital. But later he limited his practice to gynecology, and for some years, up to the time of his death, he was gynecologist to the Presbyterian Hospital. In a similar role he was on the staff of the Pennsylvania Epileptic Hospital and Colony Farm, and, in addition, was consulting surgeon to the Woman's Hospital. He was at one time president of the Philadelphia County Medical Society, and had also been president of the Obstetrical Society. For many years he was a Fellow of the Academy of Surgery. His interest in the scientific as well as the private business of the College was manifested by his frequent attendance upon the meetings. At various times he read papers, reflecting upon him much credit.

Dr. James W. Holland, elected a Fellow on December 2, 1885, died, in his seventy-third year, on February 10, 1922. For many years he filled the chair of medical chemistry and toxicology and was Dean of the Faculty of Jefferson Medical College. For some time previous to his death he was emeritus professor. A Fellow of the College has been appointed to prepare a memoir of Dr. Holland, which will be presented in the near future.

Dr. George M. Gould, elected a Fellow on May, 1897, died on August 8, 1922. He was born at Auburn, Maine, in 1848. Dr. Gould was an ophthalmologist of prominence, a frequent contributor to the literature of his specialty, for a number of years editor of the *Medical News* and the author of a very excellent and successful *Medical Dictionary.*

Dr. George A. Cameron, a native of Cecil County, Maryland, was elected a Fellow in January, 1905, and died in the fifty-ninth year of his age, on August 25, 1922. He was a well-known and successful general practitioner, and for a number of years was physician to the Germantown Hospital.

Dr. Alexander R. Craig, elected a Fellow on January 6, 1909, died on September 2, 1922, at the age of fifty-four. He was born at Columbia, Pa. Graduating in medicine at the University of Pennsylvania, he remained for several years in Philadelphia practising his profession. Later he moved to Chicago, retired from practice and became secretary of the American Medical Association, which office he filled with ability up to the time of his death.

One Fellow resigned during the year while 11 Fellows were added by election—a net gain of 4. One Associate Fellow was added by election to the Foreign list.

On the roll of the College on December 1, 1922, the Secretary tells us, there were:

> 486 Fellows; 20 Associate Fellows (13 American and 7 Foreign); 6 Corresponding Members. Total, 512.

Of the 486 Fellows, 46 are now non-resident, leaving 440 as the number residing in Philadelphia, or in its immediate vicinity in Pennsylvania.

Attendance at Meetings. The attendance of Fellows at our meetings is not what might be wished for, the average for the last year being only 42 for each meeting—the same number as in the previous year—less than 1 in 10 of the Fellows living in or near Philadelphia. At the part of the meetings devoted to private business the average is even smaller.

That this—shall it be called apathy—as to attendance upon the meetings is not confined to the older Fellows, but is evident notably among those recently added to our number, has been observed by the Secretary, in looking over the signatures of Fellows attending the meetings. It seems that in the last five years 65 new Fellows have been elected. During that period 45 meetings were held. Of these 65 Fellows, one-fifth are not credited with being present at a single meeting; 23 have been present but once; and of the entire number only 3 have attended more than 10 meetings.

Doubtless the interest of many of the recently elected Fellows is centered upon one or another of the Sections. But surely our younger Fellows should appreciate the privilege of taking part in the scientific business of the sessions of the College itself, and the responsibility of each one doing his share in the transaction of the private business of this great institution. The private sessions are brief; business of importance must often be transacted, and a fuller attendance of the Fellows to share in these proceedings is earnestly bespoken.

In a final word, I would express the grateful appreciation of the College of the continued efficiency with which the Librarian and Superintendent, Mr. Charles Perry Fisher, and the Clerk, Miss Zelner, have discharged their duties.

THE RELATIONSHIP OF EXCESS OF URIC ACID IN THE BLOOD TO ECZEMA AND ALLIED DERMATOSES[1]

BASED ON AN ANALYSIS OF OVER TWO HUNDRED CASES

By JAY F. SCHAMBERG, M.D.

AND

H. BROWN, B.Sc.

The present study was undertaken for the purpose of shedding some light on the etiology of diseases of the skin of obscure origin. It was soon found that special interest was attached to the findings in eczema. For this reason a special study was made of this disease, which comprises over one-half of the analyses made.

Cases of eczema constitute almost one-third of the cases of diseases of the skin. This disease commonly causes keen distress by intolerable itching. Sleep is interfered with, the nervous system greatly disturbed and life often made almost unbearable. The causative factors in the production of eczema have for a century been obscure. All sorts of hypotheses have been advanced, advocated, discussed, and most of them have been discarded to make way for new suppositions. The Vienna school, under the leadership of Hebra, held that the dominant causes of eczema were local, whereas the French school conjured up hereditary or acquired diatheses to explain the origin of this disease. It must be recognized that eczema is not a simple entity, but a type of reaction in the skin possessing certain clinical features. These features are not sufficiently distinctive to enable one to distinguish

[1] Read January 3, 1923.

the etiological factors involved. It is often impossible to differentiate between a chronic dermatitis due to local irritants and a true eczema of constitutional origin. There is an almost general agreement today concerning some causes of eczema. It is acknowledged that among etiological factors may be classed intestinal toxemia, protein sensitization (both food and other proteins), endocrine disorders, disturbances of the nervous system, hyperglycemia and other conditions. The older writers, particularly among English dermatologists, laid considerable stress on the association of gout and eczema. The urinary analyses and the laboratory data on which this origin of eczema was predicated were, however, regarded as so loose and unscientific as to lead to a progressively growing incredulity concerning this alleged relationship. In four out of six American treatises on dermatology published within recent years no mention of gout or uric acid in relation to eczema is made, and scant attention is paid to renal insufficiency.

Findings in Chemical Studies of the Blood. As our blood analyses accumulated, it became evident that in the group of cases studied by us the blood sugar was almost invariably within normal limits. The average content of sugar in 166 cases was 101 mg. It varied between 65 mg. and 160 mg. per 100 cc of blood. In 2 cases—a case of eczema and a case of psoriasis—it was 160 and 146 mg. respectively, but these figures, although above the average, were possibly not beyond normal limits. In only 1 case was the sugar content of pathological significance. This was in a woman, aged seventy years, who had had generalized itching for twenty years and who presented an erythematous eczema of the legs with vulvar and vaginal pruritus. She had 273 mg. of sugar per 100 cc of blood. The urine was free from sugar, but there was a history that glucose had been found in the urine on former occasions. On a diabetic regimen the pruritus promptly improved almost to the point of disappearance.

Uric Acid. In the course of our studies it soon became evident that the most significant finding was a variation from the normal of the uric acid content. It is, of course, a matter of the

greatest importance to possess an accepted standard of the maximum normal amount of uric acid in the blood.

In 1913, Folin and Denis[1] wrote that the normal uric acid content in healthy adults was usually between 2 and 3 mg. per 100 cc, but that strictly normal persons might have as low as 1 mg. and as high as 3.5 mg., the differences probably depending in part on dietary factors. Nine years later Folin[2] writes: "The maximum normal figure for uric acid may be perhaps given as 3 mg. per 100 cc."* Other investigators have given estimates of the normal limits, which are presented in the subjoined table:

TABLE I.—NORMAL VALUES ON HUMAN BLOOD IN MILLIGRAMS PER 100 CC

Non-protein nitrogen.	Urea nitrogen.	Uric acid.	Sugar.	Authority.
30–45	15–25	1.0–3.5	50–120	Gettler and Baker: Jour. Biol. Chem., 1916, xxv, 211.
22–37	12–27	0.7–3.7	95–110	Folin and Denis: Jour. Biol. Chem., 1913, xiv, 29.
30	...	...	...	Greenwald: Jour. Biol. Chem., 1915, xxi, 61.
19–39	6–20	...	100–110	Bang: Biochem. Ztschr., 1915, lxxii, 104.
25–28	...	...	50–150	Taylor and Hutton: Jour. Biol. Chem., 1915, xxii, 63.
23–44	12–27	...	...	McLean and Selling: Jour. Biol. Chem., 1914, xix, 31.
25–30	12–15	1.0–2.0	...	Meyers and Fine: Post-graduate, N. Y., 1914–1915.
				Hohlweg: Med. klin. Wchnschr., 1915, p. 331.
	10.8–25.2	...	...	Schwartz and McGill: Arch. Int. Med., 1916, xvii, 42.
		2.5	...	Moose and Zondek: München. med. Wchnschr., 1915, lxii, 1110.
			90–110	Lewis and Benedict: Jour. Biol. Chem. 1915, xx, 61.
			90–130	Michaelis: Biochem. Ztschr., 1914, lix, 166
			40–120	Strouse: Bull. Johns Hopkins Hosp., 1915, xxvi, 211.

* In a private communication received by us in April, 1923, from Dr. Folin, he writes: "The upper normal values for uric acid are probably a little higher than we have thought."

In order to check our results, we examined the blood of 17 presumably normal persons. It so happened that these were all young adults varying in age between eighteen and thirty-five years. The uric acid content varied between 1.3 and 3.5 mg. and averaged 2.5 mg.

TABLE II.—BLOOD VALUES FROM NORMAL PERSONS*

No.	Name.	Age.	Weight in kilograms.	Non-protein nitrogen.	Urea nitrogen.	Sugar.	Uric acid.	Creatinin.
1	H. R.	24	70.0	30	13	104	2.0	1.4
2	J. K.	19	61.4	30	13	110	1.9	1.3
3	W. B.	21	61.4	26	13	110	1.8	1.4
4	H. B.	25	61.4	31	15	93	2.2	1.5
5	A. K.	30	86.4	34	17	109	3.5	1.4
6	B. F.	23	61.8	27	13	120	1.9	1.6
7	J. S.	..	..	31	10	66	3.1	1.3
8	J. T.	35	70.0	36	18	107	3.4	1.0
9	J. H.	22	66.8	35	12	101	2.3	1.5
10	Miss W.	22	56.4	35	10	96	2.8	1.1
11	Miss M.	18	59.1	36	14	99	2.8	
12	Miss B.	18	57.7	35	13	105	3.2	
13	Mrs. J.	26	56.8	31	14	100	1.8	1.3
14	Miss R.	25	54.5	37	16	103	3.1	1.5
15	Miss K.	...	..	38	18	110	3.0	1.2
16	J. M.	22	66.0	33	13	96	2.9	1.1
17	Miss S.	32	..	37	18	102	1.3	1.3

* Seventeen cases between the ages eighteen to thirty-five years; average uric acid, 2.5 mg.

Of this number one had 3.5 mg.; one, 3.4 mg.; one 3.2 mg.; and two, 3.1 mg. It is not known that all of these subjects were entirely normal, but they were taken at random among laboratory workers and presumably were normal.

In patients presenting themselves with various dermatoses, different findings are encountered. Of 163 patients, 88, or 53.6 per cent, had more than 3 mg. of uric acid; 50 patients, or 30.5 per cent, had 4 mg. or more; 18 patients, or 11 per cent, had 5 mg. or more; and 7 patients, or 4.6 per cent, had 6 mg. or more.

Of 164 patients, 90 had eczema; of these, 60, or 66.6 per cent, had over 3 mg. of uric acid per 100 cc of blood; 35 patients, or 39 per cent, had 4 mg. or more of uric acid; 15 patients, or 16.6 per cent, had 5 mg. or more, and 6 patients, or 6.6 per cent, had 6 mg. or more.

It is thus seen that the incidence of hyperuricemia is higher in eczema than in other dermatoses. If it should be held that 3.5 mg. of uric acid is within normal limits, then computation discloses the fact that 45 or 50 per cent of the patients had a higher uric acid content than this amount.

Effect of Age on Uric Acid Content in Blood. There can be no doubt that age has an important influence on the amount or uric acid in the blood.

Ninety-five cases of eczema and eczematoid conditions were studied. If these are classified according to age decades it is seen that there is a gradually rising incidence in the uric acid figures (Tables III, IV and V) from twenty up to seventy years. If we compare the non-eczematous dermatoses with the cases of eczema a striking contrast in the curve is seen. The average of the various age decades comes close to normal figures and does not exhibit any rising curve.

TABLE III.—AGE INCIDENCE IN ECZEMA*

11 to 20.	21 to 30.	31 to 40.	41 to 50.	51 to 60.	61 to 70.	71 to 80.
2.0	5.4	3.3	2.2	3.3	4.4	3.3
4.6	2.7	3.3	3.9	4.8	5.8	2.2
3.1	2.8	5.5	2.9	2.7	5.6	4.3
2.4	2.4	3.3	2.4	4.7	4.0	3.5
2.1	2.2	1.8	4.1	3.8	3.2	
2.9	4.0	2.2	5.3	3.8	3.9	
3.7	2.1	3.6	6.4	2.4	2.6	
	2.8	2.8	3.6	2.5	3.9	
		3.9	4.8	3.4	6.2	
		2.9	3.1	3.6	5.0	
		4.3	4.9	3.1	4.8	
		2.6	3.4	4.2	6.2	
		2.7	2.4	1.6	2.3	
		3.4	2.3	5.2		
		3.2	3.1	6.3		
			4.5	3.5		
			2.2	5.1		
			4.9	2.9		
			4.0	3.4		
			4.9	2.8		
			4.0	6.3		
			4.1	3.9		
			5.9	2.6		
			3.0			
			4.3			
2.97	3.005	3.02	3.86	3.86	4.45	3.33

* Tables III, IV and V and the chart do not include the entire 200 cases studied: they were completed prior to a number of the later analyses.

TABLE IV.—DERMATOSES EXCLUDING ECZEMA: AGE DECADES

11 to 20.	21 to 30.	31 to 40.	41 to 50.	51 to 60.	61 to 70.	71 to 80.	81 to 90.
2.5	2.3	2.3	2.3	3.5	3.3	4.7	1.8
4.4	2.8	3.2	2.8	2.7	2.1	4.7	
2.6	2.4	2.8	2.3	2.7	5.2		
	3.3	2.5	3.3	4.7	3.0		
	2.8	6.6	3.0	3.9			
	3.0	2.6	3.1	3.9			
	1.7	2.8	4.0	2.5			
	4.0	2.2	3.2	3.3			
	3.8	2.2	3.1	3.1			
	3.1	5.0	2.6	3.8			
	4.1	3.5	2.2	3.1			
		4.3	4.6	2.4			
		2.9	4.4	4.2			
		5.0	2.8	2.9			
		3.1	2.3	4.0			
		4.8	4.0				
		4.7	4.0				
		2.2	4.5				
3.2	3.03	3.48	3.25	3.38	3.4	4.7	1.8

TABLE V.—EFFECT OF AGE ON URIC ACID CONTENT OF BLOOD

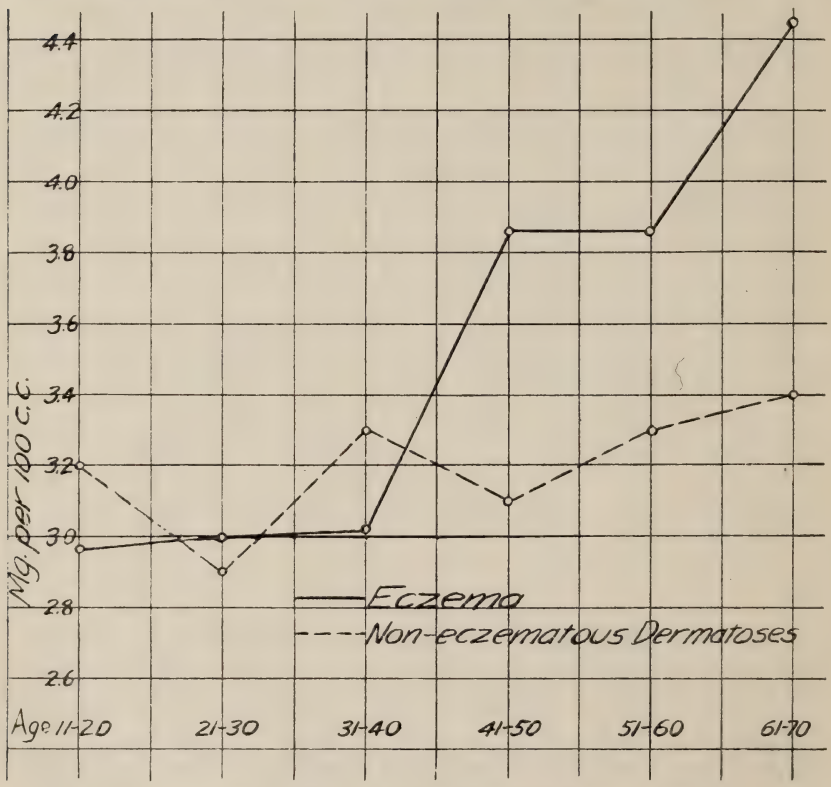
Non-eczematous dermatoses.			Eczema.		
Age.	Mg.	No. of cases.	Age.	Mg.	No. of cases.
11 to 20	3.2	3	11 to 20	2.97	7
21 to 30	2.9	10	21 to 30	3.005	8
31 to 40	3.3	13	31 to 40	3.02	15
41 to 50	3.1	16	41 to 50	3.86	25
51 to 60	3.3	12	51 to 60	3.86	23
61 to 70	3.4	4	61 to 70	4.45	13
			71 to 80	3.33	4

In the group designated eczema are unquestionably included a considerable number of cases which are eczematoid in nature, but doubt exists as to whether they constitute cases of genuine eczema. In many of these cases there are normal blood uric acid values. We have included them in order not to be guilty of any manipulation of the statistics.

For the most part, young patients with eczema have a normal amount of uric acid, but there are several notable exceptions to this statement.

Pruritus. In several of our cases of generalized pruritus in young persons there have been high uric acid values in the blood; in 1 there was 5.4 mg. and in another 4.7 mg. These cases are of particular interest.

Psoriasis. Most of the patients with psoriasis had normal uric acid figures. In a few persons past middle age the figures were high, but we believe that this was due to the coëxistence of a renal condition and that it did not bear any causative relation to the cutaneous disease. The average uric acid in 14 cases of



Effect of age on uric acid content of blood.

psoriasis was 3.2 mg. per 100 cc of blood. The average age of these patients was forty-six years.

Influence of Sex on Uric Acid in Blood in Cases of Eczema. The average blood uric acid for male patients with eczema in our series was 4.16 mg. per 100 cc, and the average for the females was 3.08 mg.

The average age of all the males whose blood was analyzed by us was 49.8 years and the average age of the females was 42.1 years. The difference in age is not sufficient to explain a difference of 25 per cent in the uric acid content. Some other factor or factors must be invoked. The fact that men are bigger eaters and larger consumers of flesh foods and alcohol than women must, we believe, be taken into consideration. The average weight of men would also be greater than the average weight of women.

Type of Patients Studied. The persons studied were all private patients from the middle and higher walks of life. They were all ambulatory or office patients and sought medical advice primarily for some cutaneous disorder. They ranged in age from fourteen to eighty-five years, but by far the largest number fell within the decades from thirty to sixty years. The blood was taken within two or three hours after a light breakfast.

The data already submitted indicate that there is a high incidence of hyperuricemia associated with eczema. This is the particular finding that we desire to present. As to the interpretation of this hyperuricemia there may be differences of opinion. There can be no doubt that some of the cases represent instances of interstitial nephritis. In cases in which the non-protein nitrogen and the urea nitrogen are high, the high uric acid figure is readily explained; but there are many cases among those studied by us in which the non-protein and urea nitrogen were clearly within normal limits.

Creatinin. It is interesting to note that in not a single patient was the creatinin in excess of the normal figure. Indeed, it varied within narrow limits, between 1 and 1.9 mg. In a case of nephritis in which the analysis was made in another laboratory, but included in our series it was 2.8 mg.

The low creatinin values are to be explained on the basis that our patients were not sick in the ordinary sense of the word, and retention of creatinin only occurs in fairly well-advanced nephritis.

TABLE VI.—AVERAGE AMOUNT OF URIC ACID IN BLOOD IN DIFFERENT DERMATOSES

	Number of cases.	Average uric acid per 100 cc of blood, mg.
Psoriasis	11	3.20
Pruritus (local and general)	11	3.20
Acne	3	3.00
Urticaria	4	3.08
Eczema	95	3.80

Case I.—K. B., aged twenty-two years, a flabby young man who was overweight, had suffered for three years with violent itching of the face and of the upper part of the torso, with secondary eczema induced in part by irresistible scratching. His sleep was greatly disturbed.

This patient had 5.4 mg. of uric acid per 100 cc of blood, with 30 mg. of non-protein nitrogen and 13 mg. of urea nitrogen. The urine was free from albumin and casts and had a specific gravity of 1.030. The blood-pressure was 130.

Under a dietary regimen eliminating foods containing purin there was an immediate and remarkable improvement in the itching and the eruption.

Case II.—W. S., aged seventeen years, who weighed 130 pounds (60 kg.), suffered from a vesiculosquamous eczema of the hands at various intervals for six years. Blood analysis revealed 4.6 mg. uric acid, 35 mg. non-protein nitrogen and 18 mg. urea nitrogen. This was the most youthful patient in our series with a high uric acid content in the blood. Eruption disappeared in a short time.

Case III.—A. McL., aged forty years, was supposedly suffering from a trinitrotoluene dermatitis of the face and hands, due to contact with this substance during the war. The entire face was red, swollen and weeping. The case proved utterly refractory to all types of local medicaments applied over a period of many months.

Blood showed the presence of 5.5 mg. of uric acid, 32 mg. of

non-protein nitrogen and 13 mg. of urea. The blood-pressure was 135 mm. The specific gravity of the urine was 1.010; there was no albumin nor casts.

The patient was placed upon a proper dietary regimen. Cured.

Case IV.—I. P. H., a man, aged thirty-nine years, with a recurring, markedly itching eczema of the legs, had had generalized outbreaks during the last three summers. The blood analysis gave the following figures: Uric acid, 4.3 mg.; non-protein nitrogen, 36 mg.; urea nitrogen, 16 mg. The father, mother and grandfather had gout. The youngest of five children had eczema. On appropriate diet and local treatment great improvement occurred.

Case V.—H. C. D., aged forty-one years, had suffered for some years from an erythematopapular eczema involving the forehead. Despite various forms of treatment, including the use of the roentgen ray, recurrences have developed. Examination of the blood disclosed the following findings: Uric acid, 4.8 mg.; non-protein nitrogen, 33 mg.; urea nitrogen, 14 mg.

An appropriate diet has been outlined for the patient, and he is at present free of eruption.

Case VI.—J. E. F., aged thirty-five years, sought medical advice for persistent areas of exfoliative glossitis of five months' duration, presenting somewhat the appearance of the "geographic tongue." His general health was excellent. The Wassermann test was negative. Examination of the blood revealed the presence of 5 mg. of uric acid, 36 mg. of non-protein nitrogen and 16 mg. of urea nitrogen. This is the only case of this character in which a blood analysis has been made by us. The etiology of this curious condition has been entirely obscure.

Case VII.—Mrs. K. G. S., aged forty-four years, had vesicular eczema on the hands and fingers and an erythematous eczema on the face. She had four or five attacks, the last of which

occurred three years ago. She had had one child. The patient felt tired and "dragged" in the mornings. Blood analysis revealed: Uric acid, 4 mg.; non-protein nitrogen, 34 mg.; urea, 13 mg. Rapid improvement occurred on a milk diet.

Case VIII.—L. S., a man, aged forty-three years, had a vesicular eczema which began on the arms one year ago and spread over the entire body. There was violent itching.

Urinalysis revealed: Specific gravity, 1.018; a trace of albumin, no casts and numerous pus cells.

Blood examination revealed: Uric acid, 4.1 mg.; urea, 16 mg.; non-protein nitrogen, 31 mg.; sugar, 82 mg. Under a low protein diet and rest the eruption disappeared.

Case IX.—J. S., aged thirty-three years, a man, had had generalized pruritus of the body for sixteen years. He weighed 168 pounds (76.2 kg.). In good health but nervous. Blood examination revealed: Uric acid, 4.7 mg.; non-protein nitrogen, 36 mg.; urea nitrogen, 16 mg.; sugar, 4.7 mg.; chlorides, as sodium chloride, 497 mg. Urine examination: Specific gravity, 1.029; no albumin nor sugar. The blood-pressure was 120.

Case X.—J. R., a man, aged twenty years, had eczema in infancy and was subject to asthma in early childhood. He now has severe eczema of the face, with marked excoriations. Various food allergens were tried, but the results were negative. The patient's general health apparently was excellent. Chemical blood analysis revealed: Non-protein nitrogen, 30 mg.; urea 14 mg.; sugar, 100 mg.; uric acid, 4.1 mg.

Inconsistencies in Blood and Urinary Findings. The non-protein nitrogen and urea may be within perfectly normal limits and yet nephritis may be present. A few such cases may be cited:

Case XI.—M. S., a woman, aged twenty-five years, had a disease which had been diagnosed as lupus erythematosus. She had nephritis in 1918 and had been confined to a hospital for some months. Examination of the urine showed a specific gravity of 1.010, 16 per cent by volume of albumin and 50,000 hyaline casts to the ounce of urine. Severe and fatal nephritis occurs in certain cases of lupus erythematosus. The blood findings were: Uric acid, 4 mg.; non-protein nitrogen, 35 mg.; urea, 16 mg.; sugar, 102 mg. It is here seen that although the patient has a nephritis the only nitrogenous element in her blood that is abnormal is the uric acid.

Case XII.—E. B., a man, aged fifty-nine years, had recurrent erythematovesicular eczema on the face, arms and legs. He selected his food and ate moderately. The urine showed a trace of albumin and hyaline casts. The blood findings were: Uric acid, 2.4 mg.; non-protein nitrogen, 36 mg.; urea, 16 mg.; sugar, 110 mg. Here the blood would indicate perfectly normal kidneys, and yet the urine would hardly support such a contention.

Case XIII.—W. L. B., a woman, aged fifty-five years, nine months ago developed an eczema which has now become a universal exfoliative dermatitis. She had albumin and casts in the urine and suffered retinal hemorrhages. The blood-pressure at one time was 200. The blood findings were: Uric acid, 5.4 mg.; non-protein nitrogen, 35 mg.; urea, 16 mg.; and sugar, 144 mg.

The non-protein nitrogen and urea were normal, but the uric acid was very high. This case emphasizes, as do many others, the fact that uric acid first accumulates in the blood as a result of damaged kidneys. At the present time the blood-pressure is 155 and there is no albumin or casts in the urine.

Instead of finding urinary evidence of kidney disease with low urea and non-protein nitrogen figures, the reverse situation may be encountered.

Case XIV.—M. W., a man, aged forty-two years, had a disease diagnosed as recurrent vesicular eczema of the hands. The blood revealed: Uric acid, 4.9 mg.; urea, 20 mg.; non-protein nitrogen, 46 mg. Urine examination revealed: Specific gravity, 1.018; no albumin nor casts.

It would seem probable that this patient had renal disease.

Other cases of a similar character might be quoted.

Apparently diet is an influenzing factor in some cases in the causation of hyperuricemia. One of the highest uric acid figures that we have observed was in a meat dealer, aged forty-two years, suffering from eczema, who was in the habit of eating from 3 to 4 pounds (1.3 to 1.8 kg.) of red meat a day. His blood showed: Uric acid, 6.4 mg.; urea, 16 mg.; non-protein nitrogen, 40 mg.

On the other hand, those who are small eaters and live largely on a non-flesh diet quite commonly have low nitrogenous blood values. A thin woman, aged eighty-five years, very careful of her diet, had only 1.8 mg. uric acid in her blood.

Discussion. A survey of the cases cited reveals the fact that a high percentage of persons suffering from certain dermatoses, particularly eczema, have a hyperuricemia.

The question of an etiological relationship between the uric acid and the associated dermatoses is a pertinent one. Gout and the uric acid diathesis were for many years invoked as the causes of many disorders, including a variety of diseases of the skin. The incrimination of uric acid in the blood as a cause of eczema is commonly found in the older text-books. Let us quote an excerpt from the work of an eminent English dermatologist writing nearly forty years ago. Tilbury Fox, in 1884, in his treatise on *Diseases of the Skin*, says:

All disorders which are connected with the excreta in the system and their circulation throughout the blood current may furnish the exciting cause of eczema. This is a clinical fact of great importance. Given the tendency to eczema, then the transmission of uric acid to the capillaries of the skin will so far derange as to aggravate, certainly, and now and again excite

an eczematous eruption. This is what is meant by gouty eczema. By securing the absence of uric acid from the circulation the eczema will often disappear and always be more amenable to treatment. The passage of uric acid through the cutaneous capillaries of an eczematous subject acts as much the part of an irritant as do some externals. A gouty state of the blood may therefore excite and modify an eczema. Some cases as I now refer to sometimes exist off and on for years and are saturated with arsenic and mercurials, but which are only relieved by recognizing the complicating item of the free production and circulation of uric acid, and instituting a régime calculated to arrest the continuance of these conditions.

Lyman, writing on gout in the *Twentieth Century Practice of Medicine,* expresses the following: "From the remotest antiquity a connection between gout and certain cutaneous diseases has been remarked by all clinical observers who have seen much of disorders that are experienced by the wealthier classes of society." Furthermore, "Eczema is frequently witnessed among the children of gouty families."

Osler writes: "Garrod and others have called special attention to the frequent association of eczema with the gouty habit."

To be sure, what we call eczema is a conglomerate concept that includes many conditions of diverse genesis, which cannot be definitely differentiated on the basis of the cutaneous phenomena alone. It would constitute valuable information if we could scientifically demonstrate some of the dominant causes of eczema. The present study suggests that hyperuricemia may be an etiological factor in the production of many eczematous conditions and allied disorders.

We are persuaded to believe that the excess of uric acid in the blood in many of the cases bears a causative relation to the accompanying eczema or allied pruritic disorder, by reason of the frequency of the association of the two conditions among the patients studied by us. We are fortified in this belief by the fact that in our experience these cases have responded more favorably and more rapidly to treatment than in previous years when the

etiological factor suggested was not suspected and the patients were not placed on an appropriate dietary regimen.

What is the significance of the excess of uric acid in the blood found by us in so large a series of cases?

Leaving out of consideration leukemia and the terminal stage of pneumonia, there are two conditions in which a high blood uric acid occurs—gout and nephritis. Without the presence of arthritic manifestations (which were absent in nearly all of our cases) or tophi, it would be hazardous to assert the belief that we were dealing with cases of hemic gout. It is quite possible that a considerable proportion of the subjects were suffering from renal change. Some unquestionably were, but it would be startling to believe that one-half of our patients with eczema—persons who were not "sick" in the ordinary acceptation of the term and who in the main had no complaint except their cutaneous distress—were nephritic. There are, it must be said, a considerable number of cases in which not only the uric acid is pathologically high, but in which the urea and non-protein also are beyond normal limits. Some of these patients exhibit albumin and hyaline casts in the urine, whereas others do not. These cases represent, we believe, beginning instances of chronic interstitial nephritis.

We have observed, however, other patients in whom the uric acid was high but in whom the non-protein nitrogen and urea nitrogen were within normal limits. What is to be said of the hyperuricemia in these cases? The urinalysis in the cases in which examination was made was normal. Brief histories of some of the cases are appended.

In a separate group must be included the cases classified in Table VIII. In most of these cases not only was the uric acid high, but the non-protein nitrogen and urea were likewise suspiciously above normal limits. These patients in the main represented the age decades from forty to seventy years, whereas the previous group contained subjects from seventeen to forty-five years of age.

Despite the fact that some of the patients in Table VIII had

negative urinary findings, they must be regarded as patients with potential renal dysfunction by reason of the nitrogenous blood values.

TABLE VII.—FINDINGS IN A GROUP OF YOUNGER PATIENTS

Name.	Age.	Uric acid.	Urea.	Non-protein nitrogen.	Blood-pressure.	Diagnosis.	Urine.
K. B. . .	22	5.4	13	30	130	Eczema	Specific gravity, 1.030; no albumin or casts.
Miss B. . .	20	4.4	13	31	...	Intestinal toxemia	
L. S.. . .	43	4.1	16	31	...	Eczema	
Mrs. K. S. G.	44	4.0	13	34	...	Eczema	
I. P. H. . .	39	4.3	16	36	...	Eczema	
A. S. . . .	43	4.9	14	30	...	Eczema	
H. C. DuB. .	41	4.8	14	33	...	Eczema	
J. R. . . .	20	4.1	14	30	...	Eczema	
Wm. S. . .	17	4.6	18	35	...	Eczema	
J. E. F. . .	35	5.0	16	36	...	Glossitis	
D. H. . .	54	4.8	14	30	160	Eczema	
A. McL. . .	40	5.5	13	32	135	Eczema	
J. S. . . .	40	4.3	18	36	...	Dermatitis herpetiformis	Specific gravity, 1.010; no albumin or casts.
S. H. H. . .	45	5.0	18	38	...	Eczema	

With respect to the younger group (Table VII) with high uric acid and low urea and non-protein nitrogen, scientific conservatism would suggest caution as to any dogmatic interpretation of the hyperuricemia. One might assume that these patients had a gouty or uric acid diathesis; on the other hand, they might be suspected of having some degree of renal incapacity. In beginning disease of the kidney, uric acid is the first nitrogenous constituent to be held back, and therefore to accumulate in the blood. Later accentuation of renal structural damage would then lead to a decreased elimination of urea and non-protein nitrogen. May both series of cases not represent merely fine gradations of nephritic change? Our knowledge of the nature of gout is still involved in obscurity. All observers admit that the kidneys play an

important role. Haig states that it is not excessive production of uric acid but faulty elimination which causes an accumulation.

Garrod expresses the same thought when he states that gout depends on a temporary or continuous decrease in the ability of the kidneys to excrete uric acid by which an overcharging of the blood with this product takes place.

TABLE VIII.—FINDINGS IN PATIENTS BETWEEN THE AGES OF FORTY-TWO AND SEVENTY-FIVE YEARS

Name.	Age.	Uric acid.	Urea.	Non-protein nitrogen.	Blood-pressure.	Urine.
D. A. P.	47	4.1	20	43	130	Specific gravity, 1.012; no albumin or casts.
C. B.	50	5.3	28	46	115	Specific gravity, 1.024; no albumin or casts.
Mr. A. W.	42	4.9	20	46	175	Specific gravity, 1.018; no albumin or casts.
H. W. H.	47	5.9	20	40	...	Specific gravity, 1.019; no albumin or casts.
Mrs. E. C.	66	5.8	15	34	185	Specific gravity, 1.026; no albumin or casts.
Mrs. B.	48	4.3	11	36		
J. K. Y.	60	4.7	15	31		
H. S. R.	62	4.5	18	37		
Mrs. B.	55	5.1	20	44	...	Specific gravity, 1.011; trace of albumin; no casts.
Dr. C.	47	4.5	19	42	...	Specific gravity, 1.019; few hyaline casts; no albumin.
C. S.	65	6.2	15	33	...	Specific gravity, 1.012; no albumin or casts; previously had casts for four or five months.
D. W. C.	60	4.2	15	40		
Mr. R.	75	4.3	13	28		
Mrs. G.	58	4.7	14	30	150	Specific gravity, 1.021; trace of albumin; few hyaline casts.
Mrs. J. M. S.	62	5.6	15	32	160	Specific gravity, 1.011; no albumin or casts.
Wm. K.	42	6.4	16	40	110	Specific gravity, 1.032; trace of albumin; many hyaline casts.

Is there in gout a specific incapacity of the kidneys to eliminate uric acid independent of permanent structural damage?

An interesting commentary on this problem is the observation that in all cases of gout that come to necropsy the kidneys are

found diseased. On the other hand, the vast majority of cases of chronic interstitial nephritis are unaccompanied by symptoms clinically recognizable as gout.

Bearing on the interpretation of our cases is the series of cases reported by Meyers, Fine and Lough.[3]

These writers reported about 30 cases in which there were high uric acid values without a corresponding retention of urea and creatinin; non-protein nitrogen was not determined. Their patients appear, however, to have been hospital cases suffering from other systemic or visceral disorders, whereas ours were patients "on their feet," and, with few exceptions, not aware that they were not well except for their cutaneous affections.

The uric acid values reported by the foregoing observers were higher than ours, but this may possibly be explained, at least in part, by a difference of technical methods employed. They state that the blood picture in early interstitial nephritis and in gout are strikingly similar, particularly as regards the increase in uric acid. They suggest that a retention of uric acid may be earlier evidence of renal impairment of an interstitial type than the classical tests of albuminuria and cylindruria.

As early interstitial nephritis and gout present blood findings which are similar, the question arises, What are the distinguishing features between the two?

Sir A. B. Garrod,[4] in a postscript to his paper, remarked that it was not his opinion that gout was entirely dependent on the power of the kidney for the excretion of uric acid; he did not think that any hypothesis could be advanced with safety. Von Noorden[5] quotes Garrod to the effect that the primary cause of the uric acid retention in gout lies in the kidney. He further says:

"Levinson and Luff showed, in a very large series of post-mortems in Denmark and England, that in cases of interstitial nephritis uric acid deposits in the articular cartilages are frequently found, although the condition was not suspected during life. Stripped of all unnecessary detail, this means that the retention of uric acid in the blood and the remaining phenomena of gout are the results of primary disease of the kidney. In

certain cases both nephritis and gout are amenable to clinical diagnosis. In other cases nephritis and, in still other cases, the uric acid diathesis remain for a long time, perhaps even up to the end of life, clinically free from symptoms and unrecognized."

He adds:

"It appears that continuous overloading of the blood with uric acid (nephritis) does not necessarily lead to gout, and, moreover, that this overloading is not followed by uric acid deposition without the accession of another, still unknown, specific gouty factor."

Folin,[2] commenting on increased uric acid in the blood in gout and in nephritis, says:

"The characteristic and extraordinary feature of pure gout is that in most cases only the uric acid is increased. Whether this peculiar fact is to be interpreted in terms of a highly selective activity and correspondingly selective deterioration of the kidneys, or whether it means that uric acid is more difficult to excrete than any other waste product, a possibility suggested by the high normal levels and the excessive variations in the uric acid content of normal bloods, is by no means clear: nor is it certain that either one of these two hypotheses can furnish an adequate explanation. In nephritis leading to true uremia there is a gradual accumulation of all the nitrogenous waste products except ammonia, and in such bloods there is no constant relationship between the increase in the uric acid and in that of the total non-protein nitrogen. But on the whole, one can say that the uric acid seldom, if ever, shows a greater percentage increase than does the total nitrogen; that the uric acid accumulation, on the contrary, is usually less pronounced. In other words, in cases of generally diminished kidney efficiency one finds no support for the idea that the uric acid retention represents any special difficulty in the process of excretion. The statement made is true at least for advanced cases of nitrogen retention. Whether it is also or equally true for the early stages representing moderate nitrogen retention is not certain. Meyers, Krauss and others have drawn the conclusion that the retention and accumulation of the uric acid precedes and exceeds that of the non-protein nitrogen in the early stages of kidney insufficiency.

Under the circumstances we would leave the interpretation of the hyperuricemia found in the series of cutaneous diseases studied by us for others to elucidate. We content ourselves with the actual findings which we have presented. A more intensive investigation, which would include the study of the purin metabolism, careful and repeated urinary examinations, blood-pressure determinations and renal function tests would doubtless shed added illumination on the subject.

Chemical Methods Employed. All blood specimens, both normal and pathological, were drawn from two to three hours after a light breakfast, and the entire analysis completed on the same day. We found sodium oxalate more advantageous than potassium oxalate as the anticoagulant, owing to its low solubility, which precludes the possibility of a large excess of the salt interfering with the subsequent protein precipitation.

The methods of analysis were, generally, those elaborated in 1919 by Folin and Wu.[6]

To 15 cc of whole blood diluted with water to 120 cc in a graduated mixing cylinder, 15 cc of 10 per cent sodium tungstate were added and the liquid thoroughly mixed. Fifteen cc of two-thirds of normal sulphuric acid were then introduced, the cylinder being constantly shaken to prevent any clumping of proteins due to excessive local acidity. After all the acid had been added the mixture was shaken for a few minutes longer. With this procedure filtration could be started at once, the filtrates being, invariably, water-clear and neutral to Congo red. The importance of having a neutral filtrate from which to precipitate the silver urate as well as the necessity of adding the acid slowly and with constant agitation to prevent adsorption of uric acid has been emphasized by various investigators.[7]

Neither the urea nor the non-protein nitrogen was determined by nesslerization. To the solutions of ammonium carbonate (obtained by the urease digestion) and ammonium sulphate (resulting from the acid digestion) were added 3 cc of 10 per cent and 3 cc of 40 per cent alkali respectively, and the liberated ammonia aërated into 5 cc portions of 0.01 normal sulphuric

acid. The nitrogen was then determined by titrating the excess of acid with 0.01 normal alkali, the indicator being methyl red containing some methylene blue as a background color. With this indicator the titration is accurate within 1.5 mg. of normal per 100 cc of blood, since a distinct color change is produced with one drop of alkali or acid. Fresh standard acid and alkali solutions were prepared each month. The exact strength of the acid was calculated from the weight of the barium sulphate obtained from 100 cc portions of the acid which were drawn from the same buret used in the subsequent titrations. The alkali, stored in a paraffin-lined bottle and protected against the action of carbon dioxide by soda lime, was titrated against the acid before each analysis and its proper factor obtained. Blank determinations were made with each new lot of reagents.

The precautions necessary in obtaining a filtrate suitable for the uric acid determination have already been pointed out. In our analyses, Benedict's phosphate standard, freshly prepared every six weeks, was employed in preference to the sulphite standard of Folin and Wu, because the more intense blue colors, obtained in the absence of sulphite, permit more accurate colorimetric comparison. Otherwise the directions outlined by Folin and Wu were strictly adhered to. During the course of our work several papers were published[8] in which the authors expressed their doubt both as to the accuracy of the uric acid determination and the non-adsorption of uric acid during the precipitation of the proteins. We therefore carefully investigated the entire subject and obtained results[9] which show that no adsorption of uric acid takes place if the tungstic acid protein precipitation is carried out as stated above, and that the Folin and Wu procedure for the determination of uric acid yields accurate results.

As a further check on our technic, analyses of normal blood specimens were made throughout the course of the work.

All figures are the averages of at least two closely agreeing results.

TABLE IX.—CASES ANALYZED

	Name.	Age.	Sex.	Diagnosis.	Uric acid.	Non-protein nitrogen.	Urea.	Sugar.	Creatin.
1	Mrs. B.	48	F.	Eczema	4.3	36	11	106	1.2
2	Mrs. D.	66	F.	Eczema	5.8	36	17	119	1.1
					6.2	44	21	113	1.8
3	A. B.	38	M.	Eczema	3.2	43	17	99	1.3
					2.7	36	18	63	1.2
4	Miss R.	18	F.	Eczema	3.7	34	19	107	1.1
5	R. W.	57	M.	Eczema	6.3	47	21	80	1.7
6	L. S.	43	M.	Eczema	4.1	31	16	82	1.7
7	Mr. H.	47	M.	Eczema	5.9	40	20	132	1.4
8	Mr. R.	62	M.	Eczema	4.5	37	18	123	1.4
9	I. W.	55	F.	Eczema	2.8	28	15	108	1.3
10	Mr. McH.	68	M.	Eczema	4.8	46	15	90	1.5
11	Mrs. M.	52	F.	Eczema	2.9	41	20	88	1.6
12	J. S. S.	56	M.	Eczema	3.4	42	15	83	1.8
13	J. S. G.	44	F.	Eczema	4.0	34	13	81	1.5
14	Mrs. B.	55	F.	Eczema	5.1	44	20	95	1.6
15	Mr. F.	59	M.	Eczema and gout	3.5	38	15	133	1.4
16	Mr. E.	50	M.	Eczema	4.9	45	25	70	1.5
17	G. A. R.	35	F.	Eczema	3.4	38	15	78	1.3
18	I. F.	49	M.	Eczema	4.0	36	21	82	1.6
19	Mr. T.	36	M.	Eczema	2.6	42	17	79	1.5
20	Mrs. K.	30	F.	Eczema	2.1	35	15	108	1.5
21	Mr. H.	60	M.	Eczema	6.3	43	21	114	1.5
22	Mr. B.	57	M.	Eczema	1.6	39	19	103	1.5
23	Mr. P.	76	M.	Eczema	3.5	40	21	104	1.4
24	D. L.	56	M.	Eczema	5.2	52	26	80	1.7
25	Mr. W.	42½	M.	Eczema	4.9	46	20	94	1.5
26	Miss J.	20	F.	Eczema	2.9	46	20	96	1.9
27	Mrs. D.	22	F.	Eczema	2.2	40	18	80	1.6
28	Mrs. K.	30	F.	Eczema (acute)	4.0	46	21	85	1.4
29	Mrs. L.	41	F.	Eczema	2.2	29	12	115	1.1
30	E. S. C.	47	M.	Eczema	4.5	42	19	93	1.5
31	C. S.	65	M.	Eczema	6.2	33	15	146	1.4
32	T. B. G.	70	M.	Eczema	5.0	35	..	104	1.97
33	D. P.	16	F.	Eczema	2.1	37	12	90	1.6
34	H. S. R.	61	M.	Eczema	3.9	37	18	105	1.5
35	Mr. C.	60	M.	Eczema	4.2	40	15	93	1.4
36	M. B. F.	53	F.	Eczema	3.6	38	19	110	1.4
37	P. H.	39	M.	Eczema	4.3	36	16	96	1.4
38	M. E. L.	61	F.	Eczema	2.6	..	..	110	
39	T. R.	34	F.	Eczema	2.9	36	15	102	1.2
40	J. H. D.	69	M.	Eczema	3.9	40	19	106	1.4
41	B. H.	11	F.	Eczema	2.4	32	14	98	1.1
42	C. K.	52	M.	Eczema	3.4	36	15	118	1.4
43	J. G. S.	57	M.	Eczema	2.5	39	19	121	1.5
44	S. B.	45	M.	Eczema	3.1	30	14	96	
45	Mrs. B.	70	F.	Eczema	3.2	38	20	273	1.7
46	J. M.	34	M.	Eczema	3.9	38	17	100	1.3
47	P. R.	75	M.	Eczema	4.3	28	13	104	1.1
48	E. C.	22	F.	Eczema	2.4	35	14	90	1.1
49	Mrs. S.	45	F.	Eczema	2.3	35	16	112	1.4
50	W. B.	67	M.	Eczema	4.0	35	15	110	1.3
51	L. H.	33	M.	Eczema	3.6	32	13	95	1.1
52	A. S.	43	M.	Eczema	4.9	30	14	106	1.4
53	E. B.	59	M.	Eczema	2.4	36	16	110	1.3
54	J. S.	43	M.	Eczema	3.4	32	13	106	1.4
55	F. C. T.	48	F.	Eczema	2.4	32	13	100	1.2
56	L. S. K.	49	M.	Eczema	3.1	33	12	125	1.3
57	E. L. S.	54½	M.	Eczema	3.8	35	14	110	1.4
58	R. E.	18	M.	Eczema	3.1	29	11	90	1.1
59	J. H.	60	M.	Eczema	3.8	37	18	94	1.3
60	H. C. DuB.	41	M.	Eczema	4.8	33	14	90	1.2
61	J. B.	42	M.	Eczema	3.6	34	15	98	1.2
62	D. A. P.	47	M.	Eczema	4.1	43	20	90	1.4
63	C. B.	50	M.	Eczema	5.3	46	28	92	1.8
64	B. B.	79	F.	Eczema	2.2	39	20	103	1.3
65	Wm. K.	42	M.	Eczema	6.4	40	16	86	1.5
66	Wm. S.	17	M.	Eczema	4.6	35	18	95	1.4
67	E. C.	60	F.	Eczema	5.8	34	15	128	1.4
68	F. G.	58	F.	Eczema	4.7	30	14	96	1.2

TABLE IX.—CASES ANALYZED—(*Continued*)

	Name.	Age.	Sex.	Diagnosis.	Uric acid.	Non-protein nitrogen.	Urea.	Sugar.	Creatin.
69	J. M. S.	62	F.	Eczema	5.6	32	15	130	1.3
70	A. I. R.	27	M.	Eczema	2.8	30	13	108	1.4
71	Dr. L.	52	M.	Eczema (mild)	2.7	54	15	102	1.5
72	P. S.	35	F.	Eczema	1.8	39	14	103	1.1
73	D. H.	54	M.	Eczema	4.8	30	14	112	1.3
74	L. K.	30	M.	Eczema	2.7	36	18	100	1.3
75	H. W.	69	M.	Eczema	4.4	39	20	96	1.5
76	K. B.	22	M.	Eczema	5.4	30	13	108	1.2
77	T. B.	39	M.	Eczema	3.3	40	19	88	1.2
78	Mrs. L.	42	F.	Eczema	2.2	30	13	98	1.1
79	A. McL.	40	M.	Eczema	5.5	32	13	101	1.3
80	M. E. S.	40	M.	Eczema (mild)	3.3	41	21	102	1.5
81	E. L. B.	44	F.	Eczema	3.9	30	14	110	1.3
82	H. T. D.	51	M.	Eczema	3.3	38	16	106	1.4
83	T. D.	19	F.	Eczema	2.0	32	13	106	1.1
84	H. T.	40	M.	Eczema	3.3	40	19	118	1.4
85	E. M. A.	26	F.	Eczema	2.0	38	12	110	1.2
86	Mrs. R.	43	F.	Eczema	3.6	34	15	110	1.3
87	H. S.	47	F.	Eczema (or dermatitis)	2.3	38	19	101	1.4
88	M. G.	23	F.	Eczema	1.8	..	12		
89	S. H. H.	45	M.	Eczema	5.0	38	18	100	1.5
90	J. J. M.	42	F.	Eczema	2.4	32	14	102	
91	L. R.	1	F.	Eczema (infantile)	2.0				
92	S. R.	32	F.	Eczema	2.7	32	15	96	1.1
93	C. L.	45	F.	Eczema	3.3	41	22	104	1.5
94	E. J. L.	67	M.	Eczema (Jan., 1921)	2.9	53	23	47	2.8
				(Jan., 1922)	6.0	38	18	62	1.7
				(Jan., 1923)	4.1	30	14	98	
95	B. C.	47	F.	Eczema	3.3	36	18	116	1.1
96	J. S.	42	F.	Eczema	3.0	..	18		
97	M. S.	30	F.	Eczema	3.3	41	22	110	1.5
98	H. J. H.	22	M	Eczema	3.0	36	18	100	
99	A. B.	38	M.	Eczema and pruritus	4.0	35	17	102	
100	S. H.	18	F.	Eczema	2.9	35	17	97	
101	J. F. K.	63	M.	Eczema	4.6	38	19	104	
102	E. S.	14	F.	Eczema	3.7	36	16		
103	M. H. S.	32	M.	Eczema	4.0	40	21		
104	C. W.	22	M.	Eczema	3.8	38	19	108	
105	H. S.	52	F.	Eczema	3.1	38	19	96	
106	S. L. S.	67	M.	Eczema	4.5	37	18	90	
107	H. M.	31	M.	Eczema	5.4	44	23	100	
108	R. J. S.	59	F.	Eczema	4.6	32	15	115	
109	M. S.	53	M.	Eczema	3.7	34	16	100	
110	D. H. F.	36	M.	Eczema	4.5	35	16	110	
111	Wm. R.	..	M.	Eczema	3.6	35	17	102	
112	Miss M.	21	F.	Eczema	2.0	..	14		
113	J. R.	20	M.	Eczema	4.1	30	14	100	
114	F. B. G.	21	M.	Eczema	2.9	31	13	92	
115	J. G. H.	40	M.	Eczema	4.1	34	16	96	
116	B. F.	38	M.	Eczema	3.5	32	16	67	
117	A. A.	65	M.	Eczema (acute erythema)	2.3	34	16	97	
118	D. G.	52	M.	Eczematoid dermatitis	2.6	35	12	102	1.4
119	Mrs. R.	61	F.	Eczematoid dermatitis	2.3	35	11	112	1.1
120	E. C.	42	F.	Eczematoid dermatitis	2.4	39	13	104	1.4
121	H. H.	45	F.	Eczematoid dermatitis	2.9	28	13	100	1.1
122	Mrs. P.	48	F.	Dermatitis	3.0	31	14	122	1.3
123	G. W. T.	30	M.	Dermatitis herpetiformis	1.7	39	20	80	1.5
124	J. S.	40	M.	Dermatitis herpetiformis	4.3	36	18	98	1.5
125	F. M.	70	F.	Dermatitis seborrheica	3.0	40	19	105	1.5
126	M. L. B.	54	F.	Neurodermatitis	3.1	37	17	98	1.4
127	P. T.	31	F.	Neurodermatitis	2.2	30	14	116	1.1
128	W. L. B.	55	F.	Exfoliative dermatitis	5.4	35	16	144	1.4
129	N. D.	60	M.	Psoriasis	2.7	27	13	113	1.3
130	A. O.	35	M.	Psoriasis	3.2	25	11	182	1.6
131	L. J.	18	M.	Psoriasis	2.5	26	9	65	0.9
132	M. G.	44	F.	Psoriasis	2.3	27	9	88	1.0
					3.3	27	14	118	1.1
133	J. A. H.	29	M.	Psoriasis	2.8	29	11	111	1.5
134	Mrs. H.	60	F.	Psoriasis	4.5	33	16	90	1.4
					3.4	42	18	93	1.6

TABLE IX.—CASES ANALYZED—(*Continued*)

	Name.	Age.	Sex.	Diagnosis.	Uric acid.	Non-protein nitrogen.	Urea.	Sugar.	Creatin.
135	R. S. I.	55	F.	Psoriasis	2.5	38	18	98	1.4
136	Mr. H.	63	M.	Psoriasis	5.2	46	23	88	1.6
137	Mrs. W.	45	F.	Psoriasis	3.2	39	14	100	1.5
138	M. D.	57	F.	Psoriasis	3.8	38	18	100	1.2
139	C. M. C.	51	M.	Psoriasis	3.1	31	13	160	1.2
140	P. R.	..	F.	Psoriasis	2.2	32	11	102	
141	E. S.	..	M.	Psoriasis	4.2	39	19	107	
142	O. C.	38	F.	Psoriasis	3.1	30	13	114	
143	C. M. C.	51	M.	Psoriasis	3.6	28	12	147	
144	J. K. Y.	60	M.	Parapsoriasis	4.7	31	15	117	1.1
145	W. M.	85	F.	Pruritus	1.8	25	14	110	1.4
146	W. H. W.	68	M.	Pruritus	2.1	32	15	113	1.2
147	Mr. McC.	60	M.	Pruritus	3.3	37	16	115	1.4
148	L. T.	37	F.	Pruritus capitis	2.3	26	11	91	1.4
149	P. A.	35	M.	Pruritus capitis	2.9	29	14	106	1.4
150	Mrs. R.	49	F.	Pruritus ani	3.1	42	20	94	1.4
151	H. G.	38	M.	Pruritus ani	6.6	42	21	90	1.5
152	Mr. L.	39	M.	Pruritus ani	3.5	29	12	102	1.1
153	J. F. S.	52	M.	Pruritus ani et hiemalis	3.1	31	10	66	1.3
					3.8	39	16	94	1.3
154	G. H. B.	51	F.	Pruritus hiemalis	2.4	38	19	98	1.3
155	D. L.	78	M.	Pruritus hiemalis	4.7	38	18	99	1.4
156	F. M.	30	F.	Pruritus ani et vulvæ	2.8	36	18	95	1.8
157	R. G.	46	F.	Pruritus vulvæ	2.3	29	13	94	
158	M. P. W.	42	F.	Pruritus generalisata	4.0	37	15	82	1.1
159	F. S. L.	52	M.	Pruritus generalisata	4.0	40	19	108	
160	J. S.	33	M.	Pruritus generalisata	4.7	36	16	101	
161	Miss K.	24	F.	Acne	3.3	34	11	71	1.4
162	I. B.	20	F.	Acne	2.6	..	12	103	
163	F. H.	23	F.	Acne	3.1	..	13	92	
164	W. S. B.	43	M.	Acne rosacea	4.6	40	18	118	1.6
165	R. S.	30	F.	Rosacea	2.4	36	10	121	1.0
166	L. B. S.	57	M.	Rosacea postulosa	2.7	46	12	123	1.3
167	B. K.	38	M.	Urticaria	2.8	40	22	70	1.4
168	I. L.	57	M.	Urticaria	3.9	33	19	91	1.5
169	Mrs. H.	48	F.	Urticaria	2.6	44	19	97	1.4
170	Mr. W.	24	M.	Urticaria (acute)	3.0	29	11	110	1.1
171	A. B.	35	M.	Defluvium capillorum	2.5	38	19	102	1.4
172	H. M.	35	F.	Defluvium capillorum	2.2	28	10	110	1.4
173	Mr. K.	43	M.	Alopecia premature	2.8	34	9	123	1.2
174	Mr. B.	61	M.	Syphilis	2.8	36	16	116	1.5
					3.8	38	16	94	1.4
175	Mrs. L.	46	F.	Syphilis	4.0	35	17	106	1.1
176	M. K.	48	M.	Syphilis latens	4.4	35	18	96	1.2
177	M. S.	25	F.	Lupus erythematosus	4.0	35	16	102	1.5
178	A. G.	21	F.	Lupus erythematosus	3.8	38	19	95	
180	A. P.	23	M.	Xanthoma	1.9	27	12	129	1.4
					2.6	31	11	134	1.3
181	Mrs. L.	44	F.	Chloasma	2.3	32	9	108	1.2
182	Miss B.	20	F.	Gastrointestinal toxemia	4.4	31	13	76	1.4
183	E. M. S.	46	M.	Keratosis of face	3.1	48	23	119	1.6
184	Mrs. O.	47	F.	Prurigo nodularis	2.2	35	17	106	1.2
185	Mrs. C.	31	F.	Gangrene of skin (acute)	2.6	38	17	109	1.4
186	J. E. F.	35	M.	Glossitis exfoliativa areata	5.0	36	16	110	1.4
187	Mr. F.	72	M.	Acute erythematous eruption	3.3	30	15	100	1.1
188	Mr. S.	46	M.	Ringworm (eczematoid)	2.8	33	15	102	1.1
189	J. H. E.	35	M.	Neurodermatitis	2.8	31	13	126	1.4
190	Mrs. L.	55	F.	Lingual hemorrhages	2.8	..	14		
191	Mr. J. B. D.	44	M.	Folliculitis of neck	3.2	32	15	110	
192	B. S.	23	F.	Fissured fingers	2.1	28	11		
193	M. F. W.	35	M.	Psoriasis	4.8	38	19	97	
194	A. K.	..	F.	Redness and scaling of face	3.2	30	11	101	
195	C. R.	48	M.	Syphilis	4.0	32	15	106	
196	C. R.	46	F.	Syphilis	4.5	47	25	102	
197	Dr. C.	34	M.	Pruritus ani	5.0	30	13	107	
108	C. S. W.	27	M.	Pruritus ani	4.1	37	18	103	
199	J. J. R.	46	F.	Leukokeratosis of mouth	2.2	38	14	97	

Reagents. Kahlbaum's urea and uric acid were used; all other chemicals were chemically pure.

Apparatus. All comparisons were made in a 5-cm. Duboscq colorimeter, by diffused daylight, by the same operator. The latter also determined all zero corrections (maximum error $< \pm$ 0.8 mm.). The two 50-cc burets used in the titrations were matched; all pipets were calibrated to deliver.[10]

SUMMARY AND CONCLUSIONS. 1. In a chemical study of the blood of over 200 patients suffering from cutaneous diseases, great variations in the uric acid content were found.

2. Over one-half of the patients had eczema, and in these an increase in uric acid beyond the maximal normal limit (3.5 mg.) was observed in 50 per cent of the cases.

3. In some cases of pruritus, both general and local, there were likewise high uric acid figures.

4. Classifying the eczema patients in age decades, it is seen that there is a rising uric acid curve from the fortieth to the seventieth year. This is not evident in the non-eczematous subjects.

5. The average uric acid content of the blood in the male subjects was about 25 per cent higher than among the female patients. The average age of the males was about seven years more than the females.

6. The blood of young patients with eczema did not, as a rule, contain large amounts of uric acid, but there were some notable exceptions.

7. The cause of the hyperuricemia is a matter for further study; we incline to the view that is to be explained on the basis of renal dysfunction.

8. Patients with psoriasis, acne, urticaria and certain other cutaneous diseases have a lower average uric acid blood content than those suffering from eczema.

9. We believe the hyperuricemia to be etiologically related to eczema and to pruritus in a considerable percentage of the cases studied.

10. Appropriate dietary measures have made many of our

patients much more rapidly amenable to treatment than patients previously treated without this therapeutic measure.

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DISCUSSION

Dr. George H. Meeker: The invitation which Dr. Schamberg gave, a moment ago, that someone else should tell the meaning of his results, cannot be accepted by me because I am frankly unable to furnish their clear interpretation. However, perhaps I can say something of value along the general problem.

First, with regard to what is the normal uric content of blood. Without question this matter is not yet decided. The most recent values given by competent authorities indicate a normal of from 2 to 4 mg. per 100 mils. In other words, the newer analyses with latest modifications of methods tend toward the fixing of higher normal figures. It is also true that even these values are still probably too low because of the fact that compounds of uric acid occur in the blood which are not reckoned in the ordinary methods of analysis as uric. Thus, in some experiments with the blood of an ox analyzed by the ordinary method, Benedict found ½ mg. per 100 mils; but after boiling with hydrochloric acid, so as to break up what was probably uric pentose compound, the uric value rose to 5 mg. per 100 mils.

Experiments are proceeding along these lines with human blood. The reported results are not conclusive, but they are sufficiently suggestive to make one think that not only are the values normally higher than is ordinarily stated, but also that there are present in blood compounds which, if broken up and an analysis then made, cause relatively high normal values for uric acid to appear. However, these facts would in my opinion in nowise invalidate the work of Dr. Schamberg and Mr. Brown, because, after all, the matter is one of comparative values. Even though the normals as determined by special methods of analysis are shown to be higher than the normals they have assumed, their relative values have all been obtained by the same method of analysis; and it would hence remain true that in their cases of eczema they have shown a hyperuricemia.

I was a little disappointed that Dr. Schamberg did not go a little further in his discussion of his findings. I thought perhaps he would say something in confirmation of the comment of Tilbury Fox that urates are actually found deposited in the skin in these eczematous cases. Was anything of that sort done by you?

Dr. Schamberg: Not as yet.

Dr. Meeker: Certainly, the idea of gout having a basis of "uric-acid diathesis" is coming to the fore again. We seem to have been ashamed of speaking of "uric-acid diathesis" in recent years. We all remember when the phrase "uric-acid diathesis" was as common in medical parlance as is the phrase "blood chemistry" today. Apparently the profession became ashamed of the term because its meaning was so vague, and its use disappeared almost entirely; but owing to the newer methods of blood analysis the phrase is having a rebirth, and the fine results of the work of Dr. Schamberg and Mr. Brown seem to be one of the exemplifications of it.

It is perhaps well for us to note in the present connection a pregnant suggestion which has recently appeared, as follows: Uric acid exists in two isomeric modifications. One modification is known as the unstable, alpha or lactam form. This form is relatively soluble. The other modification is known as the stable, beta or lactim form; and this form is relatively insoluble. Through specific enzyme action the lactam form may change to the lactim form. The lactam form, owing to its solubility, is the form by which uric acid is ordinarily excreted. Let there be, however, any body locus wherein there chances to be, from abnormal causes, a supply of the enzyme which changes the lactam to the lactim form, then the lactim form will there be produced and deposited.

Dr. Schamberg has spoken about the diet; presumably purin-free diet, such as milk and eggs. Now we cannot hope, if the above suggestion indicates the truth, that a mere exclusion of purin from the diet is certainly going to prevent uric deposits. Uric acid is not simply a catabolic product from nuclein or purin precursors in the food. Without doubt there is also an anabolism by which it is produced. That is, uric acid is produced and nucleins are produced in the body in the absence of such substances from the food. For example, growing infants may exist upon milk diet alone, which is a very good purin-free diet, yet infants have a notable production of nuclein in their growth. No doubt the regulation of the diet is an aid in preventing urate deposits, but we cannot predict that the mere exclusion of purin from the diet is necessarily going to prevent such deposits. The subject is one that could be discussed at much greater length, because it is being very actively investigated at the present time, but I feel that I have already occupied too much time, and will cease at this point.

Dr. O. H. Perry Pepper: It has been only for ten years that accurate methods for the estimation of uric acid in the blood have been available, but today such methods so exist, but only if performed by experienced chemists. Garrod required several hundred cubic centimeters, or, as he puts it, "two pounds" of blood for his "estimations" of the uric acid. Today, by the newest and best method, that of Benedict, only 0.5 cc is needed, and in expert hands great accuracy can be obtained. During the past ten years improvement in method has followed improvement with such rapidity that one scarcely has known what figures to accept or what method to follow. Today, however, the method seems almost established and satisfactory, and in every way is available for application to the routine study of patients in diagnosis and research. Still, unfortunately minor variations in technic exist and differences of opinion. For example, in the studies just reported whole blood was employed, while Folin has recently written that "*values obtained on whole blood are less convincing than those obtained on plasma.*" But this is a small matter and does not militate in the least against the acceptance of such thoroughly controlled studies as those Dr. Schamberg and Mr. Brown have reported.

In brief, they obtained normal uric acid figures in normal bloods, and in a large percentage of cases of eczema they obtained abnormally high figures, sometimes associated with high figures for total non-protein nitrogen and for urea, but in many instances the uric acid was the only fraction found to be increased.

It is this latter group which is the more significant. The finding of an increase in both urea and uric acid strongly suggests an inadequacy of

renal function, but the finding of a high blood uric acid alone in a group of allied cases is far more important and interesting. The conditions in which the uric acid alone of the fractions of the non-protein nitrogen is known to be increased are few—gout, leukemia, possibly in some forms of nephritis, after the crisis in pneumonia and after a high ingestion of purin-containing foods. It is obvious that the patients studied by Dr. Schamberg did not have true gout or leukemia or pneumonia: That such a large number of instances of even early kidney disease should occur in apparently healthy individuals and without other evidence of impaired renal function than an increase in the blood uric acid seems at best improbable. In fact, there is little more than theory to support the view that there is a type of kidney disease, the first evidence of which is a failure of elimination of uric acid. It is an attractive theory but not founded on many facts, any more than is the older suggestion that gout was merely an evidence of kidney disease.

When we consider a dietary explanation of high blood uric acid it seems unlikely to apply in these cases reported tonight. It is true, however, that while the samples of blood were collected three hours after a light breakfast, no mention is made of the types of diet during the three or four days previous, but it would take a markedly purin-rich diet to bring about these figures. It is true that a purin-rich diet will raise the blood uric acid and that this increase will lag over several days after the diet has ceased. This explanation seems improbable here unless it accidentally happens that some dietetic treatments of these cases of eczema resulted in the demonstration of a diet with a high content of purin.

None of these criticisms which I have hinted at seems to me to affect in the least the validity or the importance of the findings that have been reported, and it seems that we may accept without question that:

In many cases of eczema there occurs an increase in the blood uric acid. Dr. Schamberg has presented the facts and has left it to us to wander in the fields of speculation. Three relationships at once suggest themselves:

1. The uricemia is the cause of the eczema.
2. The eczema is the cause of the uricemia.
3. Both eczema and uricemia are results of some common cause.

If premise one is true, and the eczema is a result of the high blood uric acid, then diet should have a marked effect therapeutically in such cases of eczema as have a high blood uric acid. Furthermore, one might find uric acid in excess in the juice from the eczematous area or perhaps in the sweat of such patients. Does uric acid applied to the skin act as an irritant?

If, however, the uric acid increase was due to the eczema itself, then

perhaps one would find high uric acid in the blood in instances of widespread eczema undoubtedly due to some external local irritant. It is known that an increased excretion of uric acid occurs following extensive burns and an analogy might be drawn, but it seems very improbable that the conditions are similar.

Finally, if we assume both eczema and uricemia to be the coincident results of a common cause, then it behooves us to search further and find that cause.

It is dangerous to reason too directly from an isolated change in the blood chemistry. Possibly a much more elaborate investigation may be needed to finally unravel the problem, an investigation with the patient under complete dietetic control and with analysis of urine for the excretion of uric acid as well as of blood; but the first step is the most difficult and most important, and it is this first step which Dr. Schamberg and Mr. Brown have taken.

Dr. David Riesman: Dr. Schamberg has placed clinicians under a heavy debt of gratitude by furnishing them with important data that will prove useful as standards in the future. The figures he and his associates have presented to us were obtained from individuals who in the ordinary sense of the term are well and healthy, thus constituting a group that usually does not come, at least in such numbers, within the purview of the clinician. The work is also interesting in that it presents a comparatively new approach on the part of the dermatologist to his problems, an approach not from the skin but from the very center of the body—from the blood. Few dermatologists have attacked their problems by way of the metabolic portal. It is, however, very proper, for skin diseases are not merely expressions of local changes. The skin, besides its excretory function, may have the function of internal secretion, influencing the other organs of the body in subtle and unknown ways.

Dr. Schamberg shows the generally higher level of uric acid in cases of eczema, and it is tempting to connect these two as cause and effect. Nevertheless, it must be remembered that there are indubitable cases of eczema due to protein sensitization, so that eczema must have other causes than a high uric acid concentration. Dr. Schamberg has raised the important question as to the relation of the high uric acid content of the blood and the kidney. Is the increase of uric acid an expression—the first and perhaps the most delicate—of renal insufficiency or deficiency, or is it due to an increased formation, such as one sees in leukemia, when much nuclear material is breaking down? I do not believe that either question can be answered with a categorical yes or no. The analogy to gout, to which Dr. Schamberg has alluded, is justified, but we do not

know the ultimate cause of the high uric acid in gout. Gout is not primarily due to disease of the kidney. I am afraid we have not passed much, if any, beyond the view long ago expressed by Sydenham, who called gout "a faulty concoction of the juices of the body." If Dr. Schamberg can follow the cases of hyperuricemia—if one may use that somewhat barbaric word—and can find that later they show, by increase in blood-pressure, by albuminuria, by high non-protein nitrogen concentration of the blood, that undoubted renal changes have taken place, then we clinicians shall admit that uric acid increase in the blood is the first discoverable expression of disease or malfunction of the kidney. At present our tests give us such assurance at a much later period than is desirable.

All in all, Dr. Schamberg has made a contribution of even more importance to internal medicine than to dermatology.

Dr. Solomon Solis Cohen: As to the relation between nephritis and gout, I recall one of the great teachers, a president of this College, J. M. Da Costa, who called especial attention to the large group of gouty cases in which, with urates or uric acid and oxalates, there was more or less frequent albuminuria, and in which casts also appeared from time to time, but which in his judgment were not clinically cases of Bright's disease or cases even of the well-known gouty kidney—the interstitial or diffuse nephritis of gout, which is not uncommon. In the instances he reported he looked upon the transitory periods of albumin and casts as evidence of irritation of the kidney by the abnormal matter excreted. Certainly, all of us who have practised long enough have seen many persons having gouty symptoms who live long lives and who during these long lives show many and varied evidences of what everyone used to term, and some of us still term, the uric-acid diathesis, but who have not shown any of the clinical symptoms of disease of the kidney. That experience is so common that it hardly calls for further comment; but it certainly opposes the idea that uricemia necessarily indicates renal disease or renal failure. They may or may not occur together. Dr. Riesman, however, has suggested a very important relation that should not be overlooked, and which may be illustrated from various angles. Eczema, which by the way I believe to be a group-name rather than the designation of any single morbid process of consistent etiology—the eczemas, as I prefer to call them—occurs very largely as hereditary and family affections, and in the heredity of these families gout is also quite common. Likewise, if one traces far enough the family and ancestral groups, we find commonly in these cases other forms of arthritism, as also glycosuria, even diabetes, and various disturbances of the autonomic nerve system, from Raynaud's and Graves' disorders to angina pectoris, hayfever, urticaria and asthma.

In all these cases there may be protein or other sensitizations—I do not think anaphylaxis the right term—but these play a limited role. They indicate one of the three main factors to be distinguished. Dr. Schamberg's observations and those of his colleagues reported tonight also help us toward recognizing one of them. These three factors, upon which I am inclined to insist with perhaps somewhat meticulous reiteration, are, first, a fundamental constitutional liability, that which has been called diathesis and for which we have as yet no better name; secondly, an excitant, which may be bacterial, chemical, traumatic, dietary, meteorological or of many other orders; and, third, a determinant—that is, some anatomical, physiologic or other influence determining localization of signs and symptoms—whether in the skin, the eyes, the respiratory tract, etc., elsewhere. In all this great group of little understood affections in which the autonomic—*i. e.*, the entire vegetative—nerve system and endocrine system are involved and in which—to add to the quotation from our predecessors—there is the generation of "peccant humors," it is needful to give attention to all of these three factors, and to endeavor to recognize them as clearly as may be in the individual case. I should like to ask Dr. Schamberg whether in any of his cases of eczema reported and studied he had observed any cases of what is now called deficient basal metabolism or any other evidences of deficient or aberrant functioning of the thyroid gland?

Dr. Schamberg (closing): Answering the last inquiry first, I would say that we did not have any basal metabolism determinations made in our cases of eczema. We have from time to time seen cases of eczema associated with hypothyroidism (in fact, such a patient was seen today). We did not in our presentation enter into a detailed discussion of the causes of eczema, because we did not want to trespass too much upon the time of the College.

Dr. Riesman alluded to cases of eczema due to anaphylaxis. If a patient presents himself with eczema accompanied by asthma or hayfever, I should suspect sensitization resulting from food, animal hair, pollen or some other protein. I think that this association is suspicious of protein sensitization. Dr. Riesman referred to the possibility of bacterial infection from the skin in eczema. The primary vesicle of eczema is amicrobic, it is sterile, but there are innumerable organisms on and in the skin, and secondary infections are common. In a moist eczema there would be more chance for absorption, but we rarely see evidences of absorption of pyogenic organisms into the system. I am afraid Dr. Riesman rather misunderstood me in regard to the relationship between gout and nephritis. I believe that the kidney plays an important role in gout, but there would

appear to be something else necessary besides a renal disturbance to precipitate gouty manifestations.

Dr. Meeker's discussion of the technic of uric-acid determinations is most pertinent to the subject. If we had used the uric-acid values obtained by the method of Benedict, to which reference was made, we would have had higher uric-acid figures to report. Mr. Brown, as a matter of fact, carried out a comparative study of the uric-acid yields with the Benedict method and with Folin's method: the Benedict method gave on an average 1 mg. of uric acid more than that of the Folin. We prefer here, therefore, to use the more conservative method.

I was very greatly interested in the statement of Dr. Pepper with respect to the significance of an excess of uric acid in the blood when unaccompanied by an increase in non-protein and urea nitrogen. I had received the impression that a material increase in uric acid was regarded as evidence of some degree of renal dysfunction. The statement made by Dr. Pepper, that he believes this view is unfounded, is a most interesting and important one. If we are not warranted in regarding an increase in uric acid without a coincident rise in the other nitrogenous elements as an indication of renal damage, it is a matter that must affect all our deductions. I do not myself feel qualified to judge of the significance of this hyperuricemia. From a general survey of the various cases, taking into consideration the uric-acid findings, the urea and non-protein nitrogen findings, the blood-pressure in some cases and the urinalyses, I had rather got the impression that perhaps in the great majority of patients the hyperuricemia was the expression of mild damage to the kidney, and that the difference between those cases in which the uric acid was alone increased and those in which the other nitrogenous elements were likewise increased was one of degree rather than of kind. I had received the impression that some of these patients under unfavorable conditions were candidates for progressive renal change. I trust I am wrong in this matter, as I am naturally interested in the welfare of these patients. It appears to me that we will have to go very much further in the study of our cases to secure an adequate light upon the question of renal damage: We should study the renal function tests, make more blood-pressure determinations and determine the chemical changes in the blood constituents under the influence of diet. We have in mind animal experiments bearing upon some of the questions to which Dr. Pepper has pertinently referred—the question whether uric acid is an irritant to the skin, whether surcharging the blood of animals with large amounts of uric acid would lead to deposition in the skin, etc. There are many interesting problems which present themselves for solution in connection with this subject, and I trust that others will interest themselves in them.

THE EARLIER METHODS EMPLOYED IN THE TREATMENT OF SYPHILIS[1]

By FRANCIS R. PACKARD, M.D.

Although Ivan Bloch[2] considered that the American origin of syphilis was proven beyond contradiction, the tendency on the part of more recent writers is to regard his reasoning as fallacious. Bloch argued that because certain documents which had been relied upon to prove that syphilis existed earlier than the return of Columbus were found to be spurious, therefore the whole case fell down. But in recent years more undoubtedly authentic evidences have been discovered, and it is now generally believed by medical historians that syphilis existed in Europe long before the year 1495, in which it suddenly sprang into such terrible prominence. The works of Buret[3] and Sudhoff[4] contain overwhelming proof that in different forms and under many names, especially leprosy, syphilis was disseminated throughout all the countries of the Old World. The phenomenon of its sudden prominence corresponds with the development of a brilliant epoch in medicine, illuminated by the labors and researches of men who were to raise medicine out of its medieval thraldom. To their more exact methods and knowledge we may attribute a differentiation of syphilis from leprosy and the supposed new diffusion of the disease may in some way be compared to the kind of loose statements made by the ignorant when they speak as though the

[1] Read February 7, 1923.

[2] Ursprung der Syphilis, 1901.

[3] Syphilis in the Middle Ages and in Modern Times, translated by A. H. Ohmann-Dumesnil, 1895.

[4] Studien z. Geschichte der Medizin, Leipzig, 1912, and many other separate publications.

recognition of the disease appendicitis was that of a new disease instead of merely the result of new knowledge and power of differentiation. Certain it is that before the year 1500 there were some 19,000 leper hospitals scattered over Europe and Great Britain which vanished completely as syphilis came to the fore.

The name syphilis was first used in connection with the disease by Fracastorio in his poem entitled, "Syphilis sine Morbus Gallicus," 1530.[1] The source from whence he derived the name is unknown. In the poem he uses it as the cognomen of one of the characters, a shepherd, who is stricken with it because of an act of sacrilege. Before Fracastorio applied the term, syphilis was known as Morbus Venereus, or more popularly was designated by the name of some nation, the Spanish and Italians speaking of it as morbus gallicus or the French disease; the French terming it the Spanish disease or the Neapolitan disease. When Europeans tired of attributing it to one another they called it the New Disease and blamed its introduction on the sailors of Columbus.

Until John Hunter, in the latter part of the 18th century, differentiated chancre from chancroid, all venereal sores or manifestations, including gonorrhea, were regarded as syphilitic and treated by the same remedial measures. Ricord[2] first established the fact that gonorrhea was a distinct disease entity.

The unfortunate predominance of quackery in the treatment of venereal diseases has been a notable fact from the earliest times to the present. To a great extent this state of affairs is attributable to the feeling of shame which the disease inspires in those who are its victims and the desire for concealment on their part. Other reasons prevailed in the 15th century. Buret[3] gives three statements quoted from Torella (1500), Wendelin Hock (1502) and Ulrich von Hutten (1519), which accord in saying that the regular physicians all recognized the failure of the measures which they adopted to combat the disease, and consequently the

[1] Hieronymus Fracastor's Syphilis, from the original Latin, St. Louis, 1911; Syphilis, or a Poetical History of the French Disease, Written in Latin by Fracastorius and Now Attempted in English by N. Tate, London, 1686.

[2] Traité des maladies vénériennes, Paris, 1838.

[3] Syphilis in the Middle Ages, translated by Ohmann-Dumesnil.

quacks came to the fore, vaunting a great many supposed cures by all kinds of bizarre treatments, advertising themselves and their wares just as they do now, and numbering their victims by thousands. A very curious fact in this regard is that the most successful quack remedies of the 16th century contained mercury, which had also been the favorite medicine against leprosy, given by fumigation or inunction.

For many generations the Arabians used mercurial salves in the treatment of leprosy and skin diseases. Roger, the great medieval surgeon of the 12th century, and Theodoric, in the 13th century, advocated their use, and Theodoric describes the salivation which followed its absorption in excess.

A poem on syphilis which, though now less widely known than that of Fracastorio, had in its time great fame, is "On the Contagious and Accursed Bubas," which was appended to "El Sumario de la Medicina," by Francisco Lopez de Villalobos, first published in 1498, thirty-two years before Fracastorio wrote his poem. Villalobos was physician to Ferdinand of Spain in 1514. He was a learned and very witty man. An English translation of the poem with extensive notes was published by George Gaskoin.[1] Villalobos mentions the treatment in vogue of salivation with mercury administered by fumigations or inunctions, only to condemn it with the greatest vigor. He used mercury in an ointment solely as an application to the buboes, not for its constitutional effect. Villalobos, after giving instructions as to diet and hygiene and the use of mild purgation, recommended bleeding from the basilic vein, followed by the administration of theriaca.

Fracastorio, in his famous poem, written in 1530, ascribed the disease to astrological agencies, and stated his opinion that it had existed in various forms on the earth before its appearance as a virulent epidemic in Europe.

The principles of treatment on which he based his treatment were the chief mainstays of the therapeutics of syphilis for some

[1] The Medical Works of Francisco Lopez de Villalobos, the celebrated court physician of Spain, now first translated, with Commentary and Biography, by George Gaskoin, London, 1870.

hundreds of years after he wrote. His precepts begin with the advice to seek a sunny, dry climate, avoiding marshy, wet places, and to take strenuous exercise in the open air, such as hunting; or agricultural occupations, ploughing or hoeing and woodchopping. He also advises dancing and playing shuttlecock and palestra. He gives minute advice as to the diet; the patient should eat no fish taken from either salt or fresh water, except in case of necessity, and then only those which are fished near waterfalls, in swift-moving water. He should eat no aquatic birds, nor the flesh of hogs, nor cucumbers, truffles, artichokes, onions, vinegar or milk, and should drink only very light wines; and if the disease occurs in the spring or autumn, he should be bled copiously from the basilic or the median vein and thoroughly purged. The patient should drink freely of decoctions containing mixtures of aromatics and bitters, of which Fracastorio gives a long list. Lemon juice is particularly salutary. Ulcers in the mouth or throat are to be destroyed with verdigris, which is also useful to destroy the pustules on the skin. Osteocopic pains are to be relieved by the use of topical remedies of a soothing nature, such as goosegrease, mastic, linseed, daffodils, elder and honey.

Fracastorio says that possibly after having exhausted this series of remedies the patient loses patience because he has failed of obtaining relief by such mild but safe measures. Then, relying upon his strength, he resolves "to turn to more energetic agents to end the matter with the hated enemy as soon as possible. Be it so! I will show you these violent and expeditious methods which can triumph in a short time, over a disease that is usually long in duration, stubborn, subject to relapses and rebellious to mild medication. But also learn the price you will have to pay for your hasty deliverance." After this rather unpromising induction Fracastorio describes first the treatment by means of fumigations composed of styrax, cinnabar, antimony and incense. He says that these fumigations should never be used on the entire body, as they are very violent and irritating and may cause respiratory difficulties and suffocation. Therefore, their action should be confined to those parts which are the seat of eruptions

or ulcers. If it is decided to use fumigations he prefers to use mercury as a basis instead of the before mentioned substances. "The action of mercury on the scourge is marvellous, either because its natural affinity for heat and cold renders it proper to absorb the devouring fire of the disease, or because its surprising density permits it to divide and to dissolve the humors for a reason that is analogous to that which gives to incandescent iron a caustic action more marked than that of a light flame; or that its mobile and penetrating molecules, apt to infiltrate themselves in the warp of the tissues, have the power of consuming even to the bottom of organs the impure yeasts of the disease; or finally, that its magic virtues are derived from some occult force whose mystery escapes us."

Fracastorio says he administered mercury in an ointment mixed with black hellebore, orris root, galbanum, asafetida, oil of mastic and oil of native sulphur: "So, without hesitation, spread this mixture on your body and cover with it your entire skin, with the exception of the head and the precordial region. Then, carefully wrap yourself in wool and tow; then get into bed, load yourself with bed-covering and thus await until a sweat bathes your limbs with an impure dew. Ten days in succession renew this treatment; for ten entire days you are to undergo this cruel trial whose beneficial effects will not cause you to wait. As a matter of fact, very soon an infallible presage will announce to you the hour of your freedom. Very soon you will feel the ferments of the disease dissolve in your mouth in a disgusting flow of saliva, and you will see the virus, even the virus, evacuate itself at your feet in a copious flow of saliva."

Thus we see that the salivation of the patient was the aim of the physician, for the system thus rids itself of the poison of the disease, and this was the fundamental principle of mercurial therapy for many generations.

Giacomo Berengaria, or Berengarius of Carpi, who died in 1550, has had his name immortalized in the pages of Benvenuto Cellini for the great success he achieved in the treatment of syphilis with mercurial inunctions. Cellini says, however, that Berengarius

left Rome in great haste, and very wisely, because his patients all relapsed, "so that he would have been murdered if he had stayed."

The mercurial inunctions of the 15th century were certainly carried out in such a manner as to ensure salivation. The patient was smeared from head to foot with a thick semifluid mixture containing the mercury, which was applied with a brush, generally by a barber-surgeon or an apothecary, called by Rabelais "graisseurs de la vérole." After the ointment had been applied the patient was put in a great tub or cask and sweated. Many of the victims died of mercurial poisoning and some from exhaustion. Paré's treatise on syphilis was based, as he states, in a foreword, almost entirely on the less-known book of his friend and fellow-worker, Thierry de Héry, which had been published at Paris in 1552.[1] He gives four methods of cure:

1. With guaiac given in decoctions.
2. By friction or rubbing with ointments, most of which contained sulphur or mercury.
3. By the application of plasters containing mercury.
4. Fumigation with cinnabar.

It should be noticed that every one of these methods was based on the production of profuse sweating, the patient in order to facilitate this being swathed in blankets in a bedroom, or in the process of fumigation in a huge sort of cask. Paré does not make any mention of the internal administration of mercury.

Shakespeare alludes to the method of curing syphilis by fumigation with mercury and to the existence of hospitals or lazar houses where such treatment was given:

> PISTOL: . . . No; to the spital go,
> And from the powdering-tub of infamy
> Fetch forth the lazar kite of Cressid's kind,
> Doll Tearsheets, she by name. . . . .
> HENRY V., Act 2, Scene 1 (75–80).

To Paracelsus (1493–1541) is generally given credit for the popularization of the internal use of mercury in syphilis. Baas[2] quotes his vigorous statement: "This poison, as you call it, has a far different and a better effect than the wagon-grease with which

[1] Traité curatoire de la maladie venerienne, Paris, 1532.
[2] History of Medicine, translated by Henderson.

in the treatment of the French disease you are so fond of smearing your patients, worse than a cobbler greases his leather." He rejected all other treatments, such as decoctions of guaiac, inunctions, fumigations, etc., stating that, "If sweating will expel the French disease, a warm oven, or even a warm skin and the dog-days, would do it."

Matthiolus (1501–1577) mentions, according to Wootton,[1] that he had known women to take a pound of quicksilver to produce abortion without producing any bad effect, and he quotes Fallopius (1523–1562) as remarking that in his time shepherds gave quicksilver to sheep to kill worms, and Brassavolus (1500–1562) as stating that he had given it to children in doses of 2 to 20 grains for the same purpose.

Sprengel says that Matthiolus gave mercury internally in syphilis.

According to Wootton the first known formula for mercury in pill form was one which Barbarossa II, a pirate king of Algiers and an admiral of the Turkish fleet under Soliman, Sultan of Turkey, sent to Francis I, of France, in the second quarter of the 16th century. It may be recalled that Francis is supposed to have contracted syphilis from La Belle Ferronière. This formula, very slightly modified, was that of the famous Belloste's pills, which came into vogue about 1700 and are still retained in the French Codex.

Next to mercury, the first syphilographers placed their faith in guaiac as a remedy. It was called "the sacred wood." Guaiac was very largely popularized by a treatise written by a layman, Ulrich von Hutten, a German knight,[2] who was afflicted with syphilis when but twenty years old. In 1519 he published his "De guiaci medicina et morbo gallico," in which he narrated his

[1] Chronicles of Pharmacy, i, 409.

[2] The College of Physicians of Philadelphia possesses three interesting copies of the most important English translation of von Hutten's treatise. Thomas Paynell, a chaplain to Henry VIII, published in 1536 his translation entitled, "Of the Wood called Guiacum that healeth french pockes, and also helpeth the goute in the feete, the stone, palsey, lepre, dropsy, fallynge evyll and other diseases," of which another edition appeared in 1540. In 1730, Daniel Turner, of London, revised and republished Paynell's translation.

experiences. For nine years he was treated with mercury, during which time he thinks the treatment only served to aggravate the disease. He finally achieved his cure by drinking decoctions of guaiac.

Fracastorio in his poem, written in 1546, speaks highly of the virtues of decoctions of guaiac, but relegates it below mercury. The root of Chinese smilax was another remedy which was highly extolled in the 15th century. Vesalius in 1546 wrote a treatise on it entitled, "Epistola de Radice Chinae." It owed its fame largely to the fact that the Emperor Charles V used it for his gout, but it seems to have finally proved as useless in the cure of syphilis as it was in the case of the Emperor's gout. Vesalius thought it very inferior in value to guaiac.

Another root soon supplanted smilax and to a certain extent has maintained some reputation as an antisyphilitic, especially among the laity, even to the present day. This was sarsaparilla. Physicians still use it as a vehicle for the administration of potassium iodide.

According to Wootton,[1] a certain Antoine Lecoque, a physician in Paris, acquired a great reputation, about 1540, for the cure of syphilis with gold: "Fallopius, Hoffmann and Pitcairn, of Edinburgh, more or less fully adopted his treatment, but the theory gradually dropped out of medical practice." Early in the 19th century Dr. Chrestien, of Montpellier, revived it and it was tried out in some French hospital with very disappointing results.

The credit for introducing the use of potassium iodide in syphilis is generally ascribed to William Wallace, of Dublin, who in his "Treatise on the Venereal Disease and its Varieties," published in 1832, recommended it in advanced cases of the disease.

[1] Chronicles of Pharmacy, i, 395.

TREATMENT OF SYPHILIS FROM THE STANDPOINT OF INDUSTRIAL MEDICINE AND PUBLIC HEALTH[1]

By W. H. MACKINNEY, M.D.

The statement has been made that the importance of a disease from the standpoint of public health varies directly as its prevalence and degree of preventability. If this is true, syphilis stands near the top of the list as a public health problem. It is unnecessary to quote statistics proving the prevalence of syphilis, suffice it to say that conservative estimates place the number of syphilitics at about 10 per cent of the population. Each syphilitic is, for a time at least, a real and dangerous menace to the health of everyone by direct or indirect contact, and further remains to the date of cure or death an important factor to be considered "a safety first" in any and all lines of industrial activities, both to the employer and to employees who are associated with him or entrusted to his care.

Let us briefly consider the various ways in which syphilis may be considered from the standpoint of its relation to public health and industrial medicine.

1. Every syphilitic presenting so-called primary or secondary lesions may transmit the disease to a non-syphilitic through the medium of the secretions arising from these lesions by direct contact. This direct contact takes place through sexual relation in about 95 per cent of cases. In 5 per cent of cases extragenital contact induces infection. In 95 per cent of extragenital infection the site of infection is the buccal cavity.

[1] Read February 7, 1923.

2. Every syphilitic who marries probably transmits the disease to the other contracting party.

3. Almost every married syphilitic woman gives birth to one or more syphilitic children.

4. Syphilis is the direct etiologic factor in many diseases resulting in permanent disability, such as myocarditis, arteriosclerosis, aneurysm, paresis, locomotor ataxia, apoplexy, only to mention a few, all resulting in more or less permanent disability to the sufferer and an enormous economic loss from the standpoint of industry, public and domestic economy and family happiness; unfortunately before the seriousness of the condition is realized and before preventive measures can be applied, industrial calamities, family misfortunes and prolonged sufferings of the innocent are entailed.

Admitting then the prevalence of syphilis and appreciating its importance in relation to public health and industrial medicine, what measures have been adopted to deal with the condition and how far have they succeeded in carrying out their purposes?

Let us understand, to begin with, that the intensive efforts of federal, state and municipal authorities have only been applied to the problem of syphilis during the past four years, and probably would not have been applied at all were it not for the fact that 5.6 per cent of the first million drafted men of our late army were found with obvious gonorrhea or syphilis, so that we must not expect, in so short a time, marked results, but having made a start, it is to be hoped that it will not need another war to convince those who are concerned in the administration of offices of public health—federal, state and municipal—that the good work begun shall be continued. As far as federal activity is concerned, the U. S. Public Health Service created a department of venereal diseases and succeeded in having Congress appropriate over $2,000,000 in 1918 to bring federal aid to states appropriating money to departments for the study and treatment of venereal diseases. This appropriation was soon exhausted, and Congress, in its wisdom, has not seen fit to appropriate any more; this has reacted to the extent that many states have been

obliged to curtail their activities. The U. S. Public Health Service has further provided two or more courses conducted by authorities of national reputation for the further education of physicians who desire to become more skilled in the treatment of venereal diseases. Both of these measures have given considerable aid in the study and treatment of syphilis. It has largely, however, devolved upon the state and municipal authorities to provide educational, legislative and medical measures to combat syphilis. Pennsylvania has, during the past four years, taken an active lead in measures to safeguard public health against syphilis, and our city provides fairly adequate facilities for the proper disposition of contagious cases.

What do these health measures consist of; what are their shortcomings, and what results have been obtained as far as it is possible to judge?

1. It has been the aim of all public health services to educate the physician, first, in clinical and laboratory methods which ensure an early and prompt recognition of primary and secondary syphilis.

2. It has been their aim to inform the laity concerning the prevalence of syphilis, the source of infection and methods of contagion and the far-reaching consequences of the disease by especially written literature, educational pamphlets, lectures and moving pictures.

3. The state and municipal health authorities have caused to be passed by their various legislative bodies and health bureaus laws and regulations which allow medical men to deal with syphilis as they would smallpox, bubonic plague, diphtheria and other contagious diseases, in that it is a reportable disease. Patients may be made to submit to adequate treatment or quarantined at home or removed to hospital quarantine, and finally even a suspect may be taken into custody and subjected to an examination when believed to be a source of infection.

What more could a physician ask in the way of assistance in dealing radically with syphilis or gonorrhea?

Finally, efforts have been made to eradicate commercialized

prostitution and vice squads have brought the clandestine prostitute or street-walker to court probation.

The state has established clinics in cities where syphilitics may receive free, or at a very nominal cost, adequate treatment. Municipalities have established clinics in addition to treat this disease. The state has required every hospital on the accepted list receiving state aid to provide facilities for the treatment of syphilis. The establishment in many hospitals of a prenatal clinic has been the means of discovering the existence of syphilis in the expectant mother, and adequate treatment during pregnancy has saved the birth of a heredosyphilitic.

It has also endorsed the advertisement and sale of especially designed prophylactic packets, the early use of which will prevent infection. Certain states have attempted to require a Wassermann test as a requirement for a marriage license.

Could any more measures be promulgated to aid in the eradication of disease?

Let us now turn our attention to the results which have been accomplished through these various agencies.

From an educational standpoint, I think we will all agree that the laity have a much better conception of the prevalence, severity, far-reaching consequences of syphilis than ever before. Syphilis is a topic of common discussion; books written in plain English tell the story of the early and late symptoms, the modes of contagion and the invalidism it entails.

The campaign of education has been a success so far and the education of the youth will result in a diminution of the disease.

The education of the physician in the early and positive recognition of syphilis has proven its value. It is certain that if syphilis is radically curable at any time it is in the early chancre stage, and if secondary lesions can be prevented and rapidly cured, the chief source of direct contagion is minimized. The physician of today who procrastinates and awaits confirmation of a diagnosis of syphilis by the outbreak of a syphiloderm is negligent to the best interest of his patient and negligent to the welfare of the public.

Education has made more physicians capable of making an early diagnosis of syphilis by careful microscopic and serologic examinations.

The various state and municipal health regulations have met with a varied degree of success. I venture to say that but a small proportion of active syphilis seen in private practice is reported, and nearly all cases reported are those treated in dispensaries, where it is incumbent on the clerical force of the clinic or hospital to make such report. Why is this the case?

1. It is a trouble to the physician to go to the City Hall and get the cards.

2. It is a trouble to keep track of the numbers under which cases are reported.

3. It requires time to fill out the card and takes a personal visit to the City Hall or a two-cent stamp to return it.

4. Few physicians will take the trouble to notify the health department that a case is not reporting regularly for treatment, and many are lost track of and forgotten as new cases appear.

5. Dispensary cases are more apt to be followed up if negligent than are cases in private, as no feelings are hurt if a dispensary case falls into the clutches of the police or health authorities; but with private cases, particularly if he owes an account, it is different.

As far as quarantine regulations are concerned it is a rare occurrence compared to the number of cases encountered, and here again it is the activity of a social worker who takes more than the usual interest in the cases coming to a clinic who now and then enforces hospitalization of a flagrant contagious case which has received but little and inadequate treatment.

As to the reporting of sources of infection in a large city, it has yielded in our hands at least very unsatisfactory results. Patients in clinics and in practice frequently give fictitious names, but when asked to tell where or from whom they contracted the disease, their answers are most indefinite. They will endeavor in all ways to evade a direct answer, under the plea of having been drunk or of fear that they will incriminate themselves, and

even when investigations are made they lead the social worker frequently to a vacant lot or a garage.

Houses of prostitution have been closed repeatedly, and if the eradication of such resorts would eliminate syphilis the problem would be a simple one; but it is not so.

The chief source of syphilitic infection, if the data obtained by interrogation of all classes of patients can be relied upon, is the girl who is commonly called a "street-walker," or the girl of apparently respectable parentage who enjoys a good time in the dance hall, on the roof garden or in the automobile.

Little success has attended any legislation designed to make sexual relations difficult or unpopular.

Lastly, from the medical standpoint, the chief advances in the treatment of syphilis from a public health standpoint have been made in procuring an adequate prophylaxis. The efficiency of a thirty-three per cent calomel ointment applied a few hours after exposure has been repeatedly demonstrated and should be urgently recommended by the physician whenever deemed advisable.

In no disease other than syphilis have we a drug so certain in its activity in removing contagious lesions as we have in salvarsan. If nothing more could be said of it than this, it is enough to justify its use in practically every case of active syphilis, and it is early recognition of the disease and prompt administration of salvarsan that has done and will do more than any other measure in safeguarding the public health against syphilis.

On the whole, my general impressions are that syphilis is on the decrease, but statistics prove that we are far from having under control this most dangerous menace and economic burden.

TREATMENT OF SYPHILIS FROM THE STANDPOINT OF OTOLOGY AND LARYNGOLOGY[1]

By JAMES A. BABBITT, M.D.

Specific involvement in the otolaryngologic field presents a varied lesion which is but an expression of the underlying systemic disease, and routine treatment for primary, secondary and even tertiary lesions of the ear, nose and throat must be considered chiefly palliative. Such treatment differs little from the customary indications for mucous surface and deeper-lying tissue change of more varied origin. Special treatments of local nature have been advocated, such as the subcutaneous pilocarpine injection, mercurial wash and organic iodine compounds, and used with varying success; but as all these vascular and tissue changes converge in one fundamental reference to the spirochæte invasion, the specialist of conscience in this field inclines to submerge all treatment, save obvious hygienic prophylaxis, in the systemic tissue.

Consensus of opinion would, however, make one important exception in this general category of cardiovascular resultant—the disordered labyrinthine function, as intimate in its relation to the fundamental specific background as focal infection to its general metabolism syndrome; and in somewhat similar analogy, Guthrie estimates 5 per cent of all syphilitics as affected in the inner ear, the receptive, not the sound-conducting apparatus. It has been affirmed that one-third of all cases of nerve deafness have a + Wassermann. If one interprets correctly the purpose of this symposium, it is appropriate to omit enumeration of

[1] Read February 7, 1923.

detailed therapeusis of ordinarily topical character and discuss indication and contraindication from this vital labyrinthine standpoint for treatment by mercury and iodine or arsenic.

Certain premises of importance might be cited:

1. The labyrinthine artery, as emphasized by Kyle,[1] is the single artery of the labyrinth and a direct branch of the basilar. The labyrinthine circulation has been elaborately studied by Escat,[2] Politzer, Shambaugh[3] and others—showing its integral relation to the central brain system. It should be susceptible to similar changes, as, for example, we find in the central artery of the retina. And branches of the labyrinthine artery supply the cochlea, vestibule and semicircular canals.

2. Syphilis appears early as an involvement of the meninges, producing thickening of surface structure with pressure on important nerves of exit at their foramina, and producing from this swelling pressure neuritis and secondary atrophy.

In corollary to above, labyrinthine involvement, perhaps minor in degree, has been coexistent with the disease in its early stages, and is often associated with polyneuritis of other cranial nerves, notably the second and seventh—though conclusive indication for treatment measured by the effect upon the eighth nerve and its cochlear and vestibular distribution is peculiarly difficult to obtain because of rather meagre investigations reported on labyrinthine integrity, both prior to syphilitic invasion and under its primary stages, with and without treatment, thus coördinating the time cycle of meningeal evidence. It is scarcely possible at this point to divorce the pathological from the therapeutic side of discussion, and such intrusion in the neuroötologic field may be more justified as it is not specially captioned in the symposium.

The parasyphilitic question becomes one of profound importance in the pathological determination for systemic medication. Indeed, the functional tests of labyrinthine integrity afford one of the earliest diagnoses of the latent and resistant spirochætemia of cerebrospinal syphilis. Swift and Ellis,[4] summarizing investigations in cerebrospinal fluids, find excess of globulin and cells in a low percentage of cases in primary syphilis, and in from 36

to 67 per cent is secondary stage. Davis,[5] however, after analyzing Willcut's[6] reports of findings in 300 syphilitic cases in Urbantschitsch's Vienna Clinic, estimates a definite reduction in bone conduction in 90 per cent of syphilis in the primary stages. Wintermute[7] again has reported the observations of Benario on 210 cases of general neuritis following salvarsan administration and 121 following mercury; 79 of the 210 salvarsan cases and 46 of the 121 mercurial were of the acoustic nerve. This indicates nearly an equal result after the administration of the two drugs.

Actual nerve degeneration for neuritis begins in nerve pathway and loss of basilar membrane structure is probably irremediable. The question arises: Can partial stage be arrested, and will the therapeutic treatment produce further loss of function?

Salvarsan and neosalvarsan, or as lately termed, arsphenamine and neoarsphenamine, quickly and effectively produce their change in the general circulation, and in this result the outer ear, nose and throat receive their merited share. They do not[6] however, so effectively reach the central area which directly contributes to internal ear changes. Many authors report sudden and increased deterioration in the distribution of the eighth nerve. Ears with non-specific labyrinthine involvement are apparently made worse and syphilitic bilaterally affected ears have been reported as made stone deaf on their administration.

The answer to this may be in the so-called "neurorecidiv" explained by the Herxheimer reaction. This reaction, an increase of local manifestations about the site of the injection, is believed to be due to liberated endotoxin upon the death of spirochætes, which carry soluble toxin, and the lighting up of these in the internal ear circulation increases round-cell infiltration in the vessel wall and registers further temporary damage. Such damage, if it be in earlier stages, may be followed by improvement if dosage be sufficient to kill all spirochætes or if repeated injections produce such result. In late cases it is reported that deafness will probably be permanent.

In view of the fact that neoarsphenamine has proven of so

much simpler administration, and is so universally administered by physicians, general and special, the writer felt it would be of value to obtain a consensus of report from leading otolaryngologists in our own country as an expression of recent opinion.

The mercurial and iodide treatment is already historical. What we desire to know is whether or not the powerful and effective arsphenamine treatment is a real aural danger. In a questionnaire these men were asked to what extent they had observed positively unfavorable results from the use of salvarsan or neosalvarsan (for surety, the older terms were used). Ninety-two replies were received from letters sent to men of particular prominence. In reply the following results were obtained: 57 per cent had obtained no unfavorable results; 8 reported no unfavorable results based upon large and apparently reliable experience. In this group 6 reported particularly favorable results; 6 reported a marked preference for KI and mercurial treatment; 23 per cent reported definitely unfavorable results. Many of these reported with detailed experience and 3 referred to similar results observed in Vienna Clinics. Some of the notes accompanying were careful, and influence conclusions below; 5 per cent declared no experience, some giving no special attention to the ear; 6 per cent carried no opinion of value. These reports are not final in any way, but reflect current opinion upon this topic so vital to our field.

In conclusion:

1. In the otolaryngologic field systemic medication is chiefly important in syphilis and special dangers of such medication may apply to the labyrinthine function.

2. The neurovascular mechanism of the eighth nerve must be considered an intimate part of the central basal nervous system and as sharing the effects of early meningeal involvement.

3. Arteriosclerotic changes, as evidenced in precautionary labyrinthine tests, deserve more emphasis as contraindication to the liberal use of arsphenamine, and the customary cardiac and nephritic contraindications apply to the internal ear as well as the general system.

4. There is natural doubt in reported cases of arsenical laby-

rinthine sequelæ as to how much labyrinthine change was preexistent.

5. In labyrinthine shock, if induced by prodigal liberation of endotoxins, subsequent improvement may not proceed beyond preëxisting change due to meningeal involvement.

6. Results from questionnaire, while scarcely giving sufficient trustworthy data for final decision, emphasize the average safety in arsphenamine administration.

7. Unfavorable labyrinthine functional tests warrant preliminary mercurial and iodide treatment, followed by arsphenamine in cautious dosage.

8. An exhaustive research in labyrinthine symptomatology during early stages of syphilis would doubtless add information to our parasyphilitic knowledge.

9. Evidence of Herxheimer reaction and liberated endotoxin have not sufficiently proven actual arsphenamine injury to the internal ear, though weight of evidence warrants its occasional assumption.

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THE TREATMENT OF OCULAR SYPHILIS[1]

By EDWARD A. SHUMWAY, M.D.

The frequency of the involvement of the ocular structures during the course of syphilis makes a thorough understanding of the proper methods of treatment of syphilis from the general standpoint of prime importance to the ophthalmologist. According to Groenouw's statistics, in 7629 patients with acquired or hereditary lues the eye was involved in 3.2 per cent, while of all diseases of the eye, syphilis was shown to be the cause in 1.9 per cent. These figures agree with those determined later by Igersheimer, but, as the latter correctly observes, this percentage gives no conception of the importance of syphilis for the pathology of the eye, as, among the total conditions treated, many cases of simple conjunctivitis, foreign bodies of the conjunctiva and cornea, etc., are included. Taking only the more severe affections, syphilis is probably the cause of from 6 to 10 per cent, and according to the investigations of Widmark and others it is the cause of 14 to 15 per cent of all blindness. One needs only to name the two conditions, atrophy of the optic nerve and parenchymatous keratitis, to realize how seriously the eye is affected by the disease, and how the whole social life of a syphilitic and his descendants may be endangered by it.

Groenouw's statistics further show involvement of the various structures of the eye as follows:

Lids and conjunctiva	40
Cornea	150
Sclera	7
Iris, ciliary body and choroid	1566
Retina	229
Optic nerve	887
Lacrimal passages	49
Orbit	8
Ocular muscles	563
Various	123
Total number of syphilitic diseases	3622

Read February 7, 1923.

From this list it is evident that every tissue may be affected primarily, with the exception of the lens and vitreous, and even these two are often secondarily involved. It will be noted that, aside from the ocular muscles, which are usually affected as a result of disease of the central nervous system, the structures most frequently involved are the uveal tract, including the cornea, iris, ciliary body and choroid, and the retina and optic nerve, the retina often being diseased as the result of primary choroidal involvement.

In taking up for discussion the treatment of syphilis of the eye, I shall limit myself to the general treatment, leaving out of consideration the local treatment, although the latter is, of course, of equal importance in some of the conditions. Of particular interest is the question of the toxicity of the various antiluetic measures for the ocular tissues, especially since the introduction of salvarsan and neosalvarsan.

In the first place, the primary lesion may occur extragenitally on the eyelids, and even on the conjunctiva (in 236 cases it was found on the lids in 209 and on the conjunctiva in 27). Usually it has been caused by kissing or, in Russia, by attempts at removal of a foreign body from the eye by means of the tongue. In the treatment of this condition, excision or radical cauterization, as occasionally practised elsewhere on the body, is not possible on the conjunctiva or lids, as the cosmetic defect would be too great and the chances of radical cure are relatively slight. In former times it was taught that treatment with antiluetics should not be taken up until the appearance of secondaries. Even as late as 1908 this was recommended by von Michel in Germany. But the present viewpoint, which would seem to be the correct one, is that immediately after the diagnosis of a primary lesion is certain an intensive antiluetic treatment should be instituted. The diagnosis may nowadays be determined positively by the discovery of the spirochete in the ulcer, and we know that since the introduction of salvarsan, energetic treatment immediately on the discovery of a genital chancre may prevent the outbreak of generalized syphilis.

In the treatment of affections of the eye which occur during the secondary and tertiary stages of syphilis, in the latter of which many of the most commonly seen forms are to be placed, the same methods are to be followed out as in handling similar stages of the disease in the general system. Since the introduction and use of salvarsan, and the necessity of development of a special technic in the administration of the drug, ophthalmologists have more and more adopted the plan of referring their patients to syphilologists for the antiluetic treatment. This is probably a distinct advantage to the patient, especially to those seen in hospital dispensaries. Formerly a patient with iritis, coming to the eye service, when relieved of the symptoms after a brief course of mercury, would disappear and neglect further treatment. Now, with the strong mental effect produced by the blood test and the salvarsan injections, patients will keep under treatment more faithfully, are more easily followed up, and stand a much better chance of thorough eradication of the disease in its early stages. This should lessen the number of cases of late syphilis in the future.

Whether or not the results of the treatment of ocular syphilis have been better since the introduction of salvarsan has long been a subject bitterly disputed. Especially is this so in one of the most common of the ocular conditions—parenchymatous keratitis. This lesion is most commonly a late manifestation of hereditary syphilis, and occasionally occurs late in the acquired disease. Its course is notoriously chronic and rebellious to treatment. Many of the best-known ophthalmologists taught and still teach that antiluetic treatment is of no avail. The use of mercury was deprecated, and the same conflict of opinions which raged formerly in respect to mercury has been fought out in recent years during the salvarsan era. Fuchs is notoriously pessimistic. Igersheimer says: "As a whole, the treatment of interstitial keratitis with salvarsan has not brought about a revolution in the earlier therapeutic results. The corneal process goes on about the same as under mercury and also probably not much different than many authors had learned to know it with tonic treatment and avoidance

of mercury. There are doubtless isolated cases which clear up unusually quickly both with mercurial treatment and also after salvarsan injections, and likely these cases are chiefly of an infiltrative character." Even after intensive combined treatment with mercury and salvarsan of the first eye, the second is almost certain to be involved, and the extreme difficulty of securing a negative Wassermann reaction is a common experience in all cases due to the inherited disease. Nevertheless, Igersheimer considers the standpoint of those who discard antiluetic treatment for a purely tonic and stimulative one as completely wrong, because the indication is to influence not only the corneal process, but, above all, the general disease underlying the corneal condition. Local treatment with antiluetic remedies (subconjunctival injections, instillations and other local applications of salvarsan) have been tried, but he considers them of no avail.

In a discussion before the American Ophthalmological Society, in 1915, Arnold Knapp reported a series of cases in which he thought the inflammatory symptoms were quickly overcome by giving the patients combined treatment with salvarsan and mercury. He thought that salvarsan seemed to act best in cases with decided inflammatory symptoms, while its action was slight in cases of corneal infiltration without vascularization. Great improvement in the general health was very noticeable. Salvarsan was not able to prevent the other eye from becoming affected, but he felt that, while the expectations for salvarsan in interstitial keratitis may have been placed too high, he did not share the belief which many hold, that it is of no value in the disease, the results being very encouraging if the treatment was persevered in.

Wilder of Chicago, was of the opinion also that the duration of the disease in many cases was distinctly shortened by the use of salvarsan. de Schweinitz agreed with Knapp that salvarsan not only shortened the duration but lessened the activity of the process, and, while he was not prepared to maintain that the method facilitated the disappearance of the exudation or interstitial deposits in the cornea, he believed that it did. He had seen no evil effects as a result of its use.

We therefore advocate strongly the combined treatment with salvarsan and mercury, giving the mercury usually in the form of inunctions, under the use of which the children often gain rapidly in weight and general health. Indeed, it has been a matter of frequent observation in our clinics that children subject to the disease, instead of being dwarfed and poorly nourished, are often well developed physically and grow into sturdy and vigorous adults. Finally, it may be added that in the parenchymatous keratitis associated with acquired syphilis the results of antiluetic treatment have been far more brilliant than in the hereditary disease. The symptoms subside more quickly and the Wassermann test becomes negative more promptly. Any treatment which hastens the subsidence of inflammatory symptoms reduces the danger of blindness from opacities of the media and should be pushed in an intensive manner.

Inflammation of the iris and ciliary body, which in between 40 and 50 per cent of all cases are due to syphilis, occur most commonly in the secondary stage of the disease and respond promptly, as a rule, to the use of mercury, so that the temptation to rely upon this remedy alone is oftentimes great. But as the general experience of syphilologists points to the importance of treatment of syphilis in the early stages, in the prevention of late manifestations of the disease the ophthalmologist should follow the general teaching; the goal should not be the immediate relief of symptoms of a local character, but the thorough eradication if possible of the constitutional infection. Under the influence of salvarsan injections the lesions disappear with great rapidity, often leaving no evidence of the attack. On the other hand, neglected cases give rise to the formation of attachments to the lens, which remain permanently and may be an indication of the true cause of a later involvement of the central nervous system where syphilis had not been suspected.

Probably the most interesting problem from an ocular viewpoint in a discussion on the treatment of syphilis in general, is in connection with affections of the optic nerve, and in the remaining portion of my time I will take up a consideration of this subject

and avoid a systematic description of the lesions of the other ocular structures. It has been estimated that in approximately 25 per cent of cases of syphilitic disease of the eye the optic nerve is involved, one-third of the cases being inflammatory and two-thirds showing atrophic conditions; and that perhaps from 5 to 10 per cent of all syphilitics will have optic neuritis or its consequences. Optic neuritis may be a primary inflammation of the nerve, as in other infectious diseases, or there may be a true choked disk, as the result of gumma of the brain and its membranes, gummatous basal meningitis, syphilitic lesions of the cerebral vessels and their sequels, bilateral syphilitic periostitis of the orbit, and internal hydrocephalus of syphilitic origin. In addition to the forms of optic neuritis which show themselves on examination with the ophthalmoscope, there are types of axial involvement of the retrobulbar, intracanalicular and basilar portions of the nerve which show no changes in the eye-grounds primarily, but later may be detected by partial pallor of the nerve, especially of its temporal half, as a result of a descending degeneration.

In the treatment of syphilitic optic neuritis, mercury has always been successfully employed. When salvarsan was introduced in the treatment of general syphilis, many cases of optic neuritis were discovered, and it was assumed they were due to an arsenical intoxication, which had undoubtedly been the case in the previous use of atoxyl. Many controversies as to the true nature of these so-called neurorecidives were carried on, and progress in the treatment of syphilis of the central nervous system was greatly retarded because of the fear of their appearance. Syphilologists found, however, on more careful examination of their cases before treatment, that the frequency of such lesions was nearly as great as those reported due to salvarsan, and fortunately it was found that additional large doses of salvarsan, administered after the development of these nerve recurrences, had a tendency to cause a disappearance of the symptoms, so that the pendulum has swung in the other direction, and nearly all ophthalmologists follow the advice of syphilologists in advocating the use of a combined treatment with salvarsan injections and mercury, obtaining

in this way as intensive therapy as possible, in order to avoid the blindness which must result from a long-continued inflammation. Salvarsan is certainly the most rapidly acting antiluetic in our possession, and usually acts most favorably in nerve lesions. There is no question, also, that lumbar puncture, which should be made to confirm the diagnosis, unless there is a suspicion of brain tumor, often has a very good therapeutic effect, not only on the frequently accompanying cerebral irritative manifestations, such as headaches, but also on the process in the optic nerve itself, often modifying it favorably and improving the functions of the nerve.

Occasionally syphilitic optic neuritis is found in infants, and this is also very happily influenced by the injection of salvarsan, the dose being proportionately reduced.

Geiringer and others call especial attention to the fact that caution must be used with patients presenting evidence of early specific meningitis or any nervous condition due to recent syphilitic invasion. The early and too frequent administration of salvarsan, in these cases, has caused death from hemorrhagic encephalitis. So that it is best to begin with a course of mercury, for at least ten days, and then start with salvarsan, in small doses, gradually increasing the dose at five-day intervals.

The atrophic conditions of the optic nerve present an entirely different and much more difficult problem, and definite conclusions as to the best method of treatment cannot be reached until we have had more opportunity of observing the results from the use of some of the newer methods, introduced in recent years.

According to Igersheimer, the common causes of optic nerve atrophy in syphilis are:

1. Disease of the eye, from chorioretinitis or other severe intraocular inflammation, in which it is often difficult to decide whether the nerve is secondarily involved or is independently diseased.

2. In gummatous disease of the orbit, by pressure and disturbances of nutrition.

3. Basal cerebral lues.

4. By pressure of syphilitic tumors on the cerebral optic pathways; also following inflammation of the optic pathways themselves, whether intraocular or retrobulbar.

5. Genuine optic atrophy in tabes and general paresis.

Treatment will depend upon the cause of the process. If the atrophy has been due to an outspoken inflammatory process, active antiluetic treatment with both salvarsan and mercury is indicated, and improvement has been noted, even in fairly advanced cases, both in visual acuity and in a widening of the visual fields. With the antiluetic treatment there may be combined tonic treatment, in the form of strychnia and nitroglycerin, and some surgeons have believed they obtained good results with negative galvanism and high-frequency currents.

In the atrophy due to tabes and general paresis the outlook is very discouraging, as the degenerative process usually continues until it is complete and blindness results. Following Alexander's opinion, it has always been taught that mercury was deleterious in optic atrophy, and rapid loss of vision has been reported after its use, although there is some question whether this was certainly due to the mercury. It was also maintained that salvarsan had a bad effect, but according to Igersheimer, it can be now held as definitely proven that salvarsan does no harm to the tabetic nerve, although he does not consider that there is any prospect of success in its employment. de Schweinitz says that there are now a number of observations on record which tend to show that neosalvarsan, if used early and while color perception is still good, represents a therapeutic agent of value in the treatment of tabetic atrophy. In more recent years, intraspinal injections of salvarsanized serum, according to the Swift-Ellis method, have been employed, and also cerebral injections have been advocated by Schoenberg and others with apparently some good results if the degenerative process is not advanced. Many neurologists, especially of this vicinity, apparently have substituted for the intraspinal injections intravenous injections of salvarsan combined with subsequent punctures of the spinal cord, as advo-

cated by Dercum. Whichever method is employed it should be given a good trial in the hope of checking an otherwise hopeless process, and the ultimate opinion as to the result achieved must be formed only after more time has elapsed and more facts secured. To this end there should be a close coöperation between the neurologist and the ophthalmologist, and a systematic ophthalmic examination of all syphilitics should be made repeatedly from the beginning of the disease.

THE TREATMENT OF SYPHILIS AS SEEN BY THE INTERNIST[1]

By THOMAS McCRAE, M.D.

The worker in internal medicine meets syphilis in all its forms, but he is particularly concerned, for the purposes of this symposium at all event, with the later stages of the acquired form and congenital syphilis. In the short time allowed it is not possible to enter into details but advisable to discuss the general principles involved.

Let us always have a clear idea as to what we are striving to do in treating the patient with syphilis. We should consider the patient as well as his syphilis. In the acute stages—so-called primary and secondary—we have to deal mainly with the spirochætal infection. Naturally the patient needs attention, but in a general way our aim is to endeavor to kill the spirochætes, and usually the patient is safe with ordinary care in giving intensive treatment. Our object is to hit the infectious agent, and hit hard. But in the later stages—the so-called tertiary and quaternary stages—the problem is a very different one. We have a patient with a chronic spirochætal infection, which may be active or quiet, and in addition some visceral damage. How much we can accomplish in ridding the patient of the spirochætes is often doubtful. We hope to do it. But care must always be exercised that we do not injure the patient, which should always be a primary consideration. The patient with congenital syphilis is perhaps between these groups, but in the late congenital cases caution is necessary. A comparison may be made with the treatment of malarial fever. In the ordinary form of infection we need but give slight consideration to the patient, as he is not

[1] Read February 7, 1923.

likely to be harmed by our treatment, but if he has "black-water fever," it is possible to do material harm by careless treatment.

The problem of treatment, therefore, falls into two parts: (1) Destruction of the spirochætes, and (2) the restoration of the damaged tissues so far as this is possible. The first involves the use of arsenic preparations and mercury; the second of iodide. There is no question of controversy between the use of arsphenamine and mercury, for both have a place. Of the arsenic preparations there seems no question that neoarsphenamine is much to be preferred to arsphenamine; in fact, it is a question if the latter should ever be used in the later stages of syphilis. My own feeling is that it should not be used, as the risks are so much greater. Of the other arsenical preparations and modifications no one seems to have proved its superiority to neoarsphenamine.

What are the special points to be kept in mind in treating the patient with visceral syphilis? The frequency of involvement of the circulatory system must be remembered. Patients with aortic or myocardial syphilitic disease may be harmed by the careless use of arsphenamine or neoarsphenamine. But more important is the possibility of syphilis of the liver. If this is present there is great danger from the use of the arsenic preparations with their affinity for the liver cells. The possibility of hepatic syphilis should always be kept in mind before giving arsenical therapy, and if there is any evidence, even slight, of disease of the liver or any form, arsenic preparations should not be used. In considering the use of mercury special attention should be given to the condition of the kidneys and care taken to do no harm to them.

The question arises as to how much we should be guided in treatment by the Wassermann reaction. It is essential to know by whom the test is done, for if in careless or incompetent hands it is worse than useless. A positive reaction is of much value, but no great weight should be given to a negative reaction, as this occurs not infrequently in visceral syphilis. The general clinical findings may be more important than the serological results. Clinical suspicion justifies treatment even if the Wassermann test

is negative. Certainly no attempt should be made to secure a negative complement-fixation test if the treatment is doing harm to the patient.

As a general rule it is best to begin treatment by giving mercury if the state of the kidneys does not prevent this. Inunctions are the most effective and injections come next. After a course of these, neoarsphenamine may be given if hepatic syphilis can be excluded. A small initial dose is advisable, say 0.2 to 0.3 gram. If this is tolerated well, succeeding doses may be given of 0.6 gram. The number must depend on the patient, but a series of four to six given once a week may be regarded as an average. Any sign of skin irritation, which may be seen first at the site of the injections, or jaundice should be a signal to stop the administration of arsenic. It is usually well to repeat the course of mercury and further administration of arsenic should be decided by the result. In any case it is good insurance to give a course of either mercury or arsenic, or both, once a year for some years.

In the cases of syphilis of the vascular system the main dependence should be placed on mercury. Neoarsphenamine should be given cautiously and in small dosage until one is sure of its being well tolerated. If there is syphilis of the myocardium it is usually wiser to trust to mercury alone. With syphilis of the lung both mercury and arsphenamine may be used fairly energetically. With syphilis of the liver, as already said, arsenic preparations should never be given. In the treatment of syphilis of the nervous system both remedies are to be employed. The special methods are familiar and do not require discussion.

Of the second object of treatment, the restoration of the damaged tissues, the possibilities, limitations and difficulties should always be considered. A damaged aorta can be restored to a very slight extent at the best; a lung with syphilitic lesions will be left with much fibrosis in healing; sclerosis in the nervous system cannot be changed, and endarteritis can be influenced but slightly in the majority of cases. The organ which shows the greatest possibility of repair is the liver. The remedy for the changes in the tissues is iodide. The dose is not to be set in definite figures. Save in

some cases of syphilis of the nervous system, my opinion is that very large doses are rarely necessary. Probably 2 grams (gr. xxx) a day do as much as larger amounts. If it is given well diluted in water or milk there is rarely difficulty in its administration. If symptoms of iodism appear an increase in the dose sometimes overcomes this difficulty. As to the duration of treatment by iodide—it can never be too long and patients should take it for some time, at least every six months.

We should always remember that we are treating a patient in addition to our effort to kill the spirochætes. The patient is the first consideration and better that he should live harboring some spirochætes than that he should suffer harm from treatment.

DISCUSSION ON PAPERS OF DRS. PACKARD, MACKINNEY, BABBITT, SHUMWAY AND McCRAE

Dr. George M. Coates: Dr. Babbitt's paper, which is the only one I shall attempt to discuss, deals largely with the subject of syphilis of the internal ear and eighth nerve, but as the title covers rhinolaryngology as well, I shall speak for a moment of lesions of the nose and throat. As is well known to all of you, the mucous membrane of the throat and mouth, and, to a lesser extent, the nose, is a favorite place for the appearance of extragenital chancres, of secondary skin lesions, modified in character by the moisture and heat, and of tertiary lesions, such as gummata. Primary lesions of the upper respiratory passages are usually easy of diagnosis if the observer is on the lookout for them, and clinical diagnosis is easily confirmed in both primary and secondary lesions by the dark-field examination. Here, as in other parts of the body, if diagnosis is made in the most early stages and arsphenamin treatment begun at once, before the disease has become systemic and before it shows a positive Wassermann, it may be aborted and permanently cured. Again, secondary lesions, of which the mucous patch is the type, yield rapidly to arsenical treatment. Gummas involving mainly the soft tissues were among the early spectacular cures reported when salvarsan first came into use, but when there is destruction of bone the cure is neither so rapid, so spectacular nor so complete and permanent. As Dr. Babbitt has said, local treatment of these conditions helps but little, except in so far as removal of necrotic structures aids in the work of repair. Mercury and the iodides are stand-

bys that for years have given satisfactory results in many cases; but as in many instances the time element is of importance—in grave stenosis of the larynx or esophagus, in threatened perforation of the hard palate and similar conditions—the more quickly acting arsenic preparation is the remedy of choice, to be followed, later, by mercury, to complete the eradication of the spirochetes, and iodide to help absorb the inflammatory products.

In syphilis of the internal ear and eighth nerve we have a somewhat different condition to deal with. The eighth is probably the most vulnerable of all the cranial nerves to the syphilitic infection, and this infection can be recognized quite early in the course of the disease. By tests of the cochlear and vestibular function the general character of cerebrospinal syphilis can often be recognized before it is manifest elsewhere. Tinnitus and impaired hearing appear frequently at quite an early stage, at times coming on gradually but often appearing with suddenness, so that there is an abrupt onset of tinnitus and profound deafness. If the specific nature of the deafness is recognized at an early stage it seems reasonable to suppose that rational treatment with arsphenamin will at least limit its progress, though it is useless to expect that nerves that have atrophied and been replaced by fibrous tissue can be restored to function by this or any other means. Whether the administration of arsphenamin can itself cause the sudden almost total deafness occasionally reported is still a moot question, as Dr. Babbitt has observed; but I agree with him that probably these cases are due to the luetic infection, possibly accentuated by suddenly liberated endotoxins. If the nerve and end-organs have not been permanently damaged, arsphenamin, by relieving them of all further infection, should give them the chance to resume function at a later period, and in fact we often see this happen. So many disappointing results have, however, followed this treatment, that I think it is now common practice for otologists to follow the administration of arsphenamin (whether or not a negative Wassermann reaction has been obtained) by the customary mercury and iodides for a long period of time, and to watch the blood and spinal fluid at suitable intervals for any recurrences. In these internal ear cases, as in the oral lesions, the time element is again essential, so that the more quickly acting arsenic preparation is used to check the grave and irreparable damage that is being done to the organ of hearing. That is all that arsphenamin is expected to do. The mercury and iodides are relied upon to restore that part of the function—hearing—that has not been permanently destroyed.

Dr. J. Milton Griscom: In syphilitic disease of the eye, salvarsan was most effective in primary and early secondary lesions, while mercury

and the iodides were more to be depended upon in the late secondary and tertiary manifestations. In most cases the combined use of both measures was necessary to effect a cure.

In interstitial keratitis attention was called to the fact that in certain cases there is a mixed syphilitic and tuberculous infection which demands that appropriate treatment be given for both underlying causes. In this disease, reference was made to the use of thyroid extract in addition to antisyphilitic treatment.

I wish to emphasize the point made by Dr. Shumway with regard to coöperation between ophthalmologist and syphilologist, since a clinical cure does not necessarily mean a complete cure, and many recurrences could be avoided if patients were referred to the syphilologist for a final survey.

Dr. A. A. Stevens: To the internist interest in syphilis centers chiefly in its involvement of the central nervous system and cardiovascular system. The neurologist is in the best position to discuss syphilis of the nervous system, both interstitial and parenchymatous. In regard to cardiovascular disease, I think the importance of syphilis as an etiological factor is still not fully appreciated. It has been shown that syphilitic lesions are present in the vessels or heart in from 6 to 7 per cent of all necropsies, and if the diagnosis is based upon histologic studies, in not less than 30 to 40 per cent of all cases in which there has been clinical evidence of lues. The point of election is the aorta, and in about 90 per cent of the cases the ascending aorta or ascending arch is chiefly involved. Unfortunately, it is not possible to detect aortitis in many cases until it has led to aortic insufficiency, aneurysm or myocardial insufficiency. Therefore, it is not possible to recognize the disease at the stage when any antiluetic treatment would be likely to be of great service. Nevertheless, I think that it is possible, in a certain proportion of cases, to detect aortitis at an early period before serious damage has been done. The chief indications are a soft, systolic murmur in the aortic area, accentuation or a metallic quality of the second sound, widening of the aorta, as shown by roentgen ray and pain of varying intensity in the region of the manubrium. At this period of the disease appropriate antiluetic treatment may arrest the process and even remove the objective signs. As regards the later lesions, I think that syphilis should be suspected whenever there is aortic insufficiency without an accompanying true mitral stenosis. The occurrence of true mitral stenosis in connection with aortic insufficiency makes the etiology doubtful, as syphilis seems to play no part in the production of endocarditic mitral lesions or of hypertensive cardiovascular disease. As to treatment, I believe that in all cases of cardiovascular disease in

which there is evidence of syphilis, antiluetic treatment should be given, provided the lesions are not so far advanced that only palliative treatment is likely to be of avail. Further, I believe that arsphenamin itself should never be used in cardiovascular syphilis, but preference should always be given to neoarsphenamin. The latter is less toxic, less powerful, and acts more slowly than arsphenamin, and therefore gives more time for repair during the removal or absorption of the syphilitic exudate. Moreover, it can be given in greater concentration, that is, in less saline fluid. The large amount of fluid required for the administration of arsphenamin may in itself be detrimental if there are any indications of cardiac insufficiency. Even neoarsphenamin should not be given, I think, until the patient has been treated for a time with mercury and iodide. Mercury is best given by inunction or by intramuscular injection. Administration by the mouth is most convenient, but it is the least eligible method, because it is the least certain in its effects. For intramuscular injection, the salicylate is a reliable preparation. It is relatively rich in mercury, and when given in oily suspension it excites very little pain, and, as shown by roentgen examination of the site of the injection, it is absorbed within a week. One mil of a 10 per cent suspension in oil may be given once a week for six weeks in conjunction with potassium iodide by the mouth. After this combined treatment neoarsphenamin may be used, but the initial dose should be very small, not more than 2 dcg. If this amount is well borne the dose may be increased to 11 or 12 dcg., provided there are no untoward effects. After six or seven weekly injections a course of mercury and iodide should again be given. In cases presenting symptoms of pronounced myocardial insufficiency it is inadvisable to use organic arsenic at all until compensation has been restored, or nearly restored, by rest and digitalis. In advanced cardiovascular syphilis, antiluetic treatment does not change the physical signs, but it may bring about a definite improvement in the patient's general condition, as shown by a gain in weight, a better color and a decrease in pain, etc.

I am fully in accord with what Dr. McCrae has said in reference to the use of arsphenamin in hepatic syphilis. Organic arsenic itself is quite capable of producing destructive changes in the liver. In early, florid syphilis, when there is a precocious involvement of the liver, organic arsenic is likely to result in harm. Even in late syphilis, I believe that mercury and iodide alone will accomplish as much in removing hepatic lesions as a combination of mercury and iodides with arsphenamin.

Dr. F. X. Dercum: I did not expect to be called upon to speak. I came to the meeting of the College with the pleasurable anticipation of being interested and instructed by a series of brilliant papers and by an

excellent discussion, and I have not been disappointed. I have cheerfully and wholeheartedly joined in the applause which the papers and discussion have elicited. The subject of neurosyphilis is a vast one. It is almost impossible to more than touch upon it in the few minutes at my disposal. As regards the nervous system, the most important thing is to distinguish between the two forms, gummatous and parenchymatous syphilis. Our clinical experience in regard to the distinction between gummatous and parenchymatous syphilis fully bears out the remarkable researches of Levaditi and Marie, made in 1919 at the Pasteur Institute, which indicate clearly the existence of two different strains of spirochetes. Personally, I am convinced of the existence of these two strains, although it has not yet been possible to differentiate them morphologically. I believe that some day this will be done. The symptoms of gummatous syphilis of the cord and brain and those of tabes and paresis are so different that clinically they cannot be included under the same caption; they are not the same disease; they are utterly different and distinct. So it is in regard to therapeutic results. I remember that in regard to the older methods of treatment our results in gummatous syphilis were sometimes marvelous. I remember, for instance, how two cases were carried into the hospital many years ago completely paraplegic and how these two patients later on walked out of the ward—not entirely cured, it is true, but wonderfully improved. They were cases of gummatous infiltration of the membranes and walls of the bloodvessels and not of disease of the nerve substance. In tabes we have to deal with a very different problem. We are confronted with tissue changes which have taken place in the substance of the spinal cord. Our object is, of course, to arrest the progress of the disease and prevent further damage, but I think we should proceed with caution as regards the salvarsan. There can be no doubt that a too intensive treatment may do harm; especially is this to be borne in mind in the case of paresis, in which, in my judgment, the treatment should be intensive only in the early stage. In the pronounced or advanced cases treatment is commonly of little avail, and a too free use of the arsenic may actually abridge rather than lengthen the life of the patient.

In tabes, too, we cannot replace the tissue, but we can reduce the pain which the patient suffers and we can lessen the ataxia. In our clinic at the Jefferson Hospital spinal drainage has proved a very valuable adjuvant in the treatment. Intradural pressure is reduced, the nutrition of the cord is enhanced by the passive hyperemia induced and the diffusion of the salvarsan and mercurials promoted. We have long ago abandoned the Swift-Ellis method, believing that such favorable results as followed the latter were merely due to the incidental drainage.

Dr. Richard C. Norris: After the excellent paper by the representative of the Public Health Service, you will understand the viewpoint of the obstetrician and his recognition of the great importance of this subject in maternity work as perhaps in no other field, and we hope, by early diagnosis and treatment, to get at the root of the matter of preventing congenital syphilis. We know the ill effects of this scourge. Twenty per cent of stillbirths and the same percentage of abortions are due to syphilis. In the old days, before these newer investigations of medical diagnosis, we found, in our clinical studies, that about 3 per cent of women coming to the Maternity Hospital were the victims of syphilis. Under newer methods of diagnosis this percentage has at least doubled. Six to 8 per cent is the average report and in some districts, especially among negroes, it is as high as 19 per cent. We cannot, for diagnosis, rely any longer upon clinical investigation alone. It is well known that many women who are pregnant have so-called "latent syphilis." Many of them will produce provocative reactions, one observer stating that 10 per cent have shown a positive reaction that disappears after labor, due to some metabolic changes in the liver or placenta and not due to syphilis. Others call these cases "latent syphilis." Nowadays it is acknowledged in prenatal work that the diagnosis must go hand-in-hand with treatment. As a matter of fact, as the technicians will tell you, we have not reached the stage of exact method. One reaction, or one choice of treatment, may be relied upon in one place and another method is preferred in another part of the world. There are many ways of confirming the diagnosis and making it more and more exact in our maternity hospitals than is ordinarily done. I am sorry to say that there is no maternity that I know of whose reports show the work done in the most exacting way. Usually one blood examination of the venous blood at the elbow and one examination of blood from the umbilical vein make the diagnosis for or against syphilis. The relative value of blood from the elbow, from the umbilical vein, from retroplacental blood, of microscopic studies of the placenta and of the fetal organs—all should be studied. Maternity hospitals have to be equipped with experts in this line to study cases from every viewpoint and routinely, and I know of no more important work in approaching the question of combating syphilis than to insist upon this kind of study and work being done. At the Nauheim Congress, in 1920, Loeser advocated compulsory Wassermanns in every pregnant woman before and after labor. We have known the value of treatment in obstetrics and we have seen remarkable results in recent years. Williams, of Johns Hopkins, reported 172 pregnancies in 49 syphilitic women. Before treatment the infant mortality was almost 60 per cent, and of these same women who received treatment in 85 pregnancies the infant mortality was reduced

to about 10 per cent. Beck reported in 17 cases receiving 5 or more injections, 14 non-syphilitic children. So that obstetricians have come to believe that the pregnant woman is unusually successfully treated and some of the most brilliant results are obtained. Whether we should rely upon mercurials or the newer arsenical preparations still is under study. There are cases in which the liver function is so badly damaged that we cannot use neoarsphenamin and we must rely upon the preparations of mercury and iodide. There are others with badly damaged kidneys in which you may choose arsenic. The results achieved with the old treatment, and particularly with the more modern methods, have been remarkable. Nowhere will you see more brilliantly than in maternity work the beneficial results of treating syphilis. I believe it offers a golden field for this work.

If 5 to 7 per cent of all pregnant women in hospital and private practice are victims of syphilis, latent or otherwise, and such practical results are obtained from scientific diagnosis and treatment, it certainly is desirable to incorporate such studies in all of our prenatal work.

Dr. John A. Kolmer: I shall devote my five minutes to a discussion of the value of the Wassermann or complement-fixation test as a serological guide to the treatment of syphilis. Time does not permit an adequate discussion of the mechanism of this reaction nor a discussion of its sources of biological and technical error, although I may say that in the last two years a great deal of new knowledge has been gained on both subjects. From the standpoint of the reaction, I believe it may now be stated that the reaction is far more specific for syphilis than a review of the literature on this subject would lead one to conclude. As far as my experience is concerned, I recognize only one other disease capable of giving a truly positive, technically correct Wassermann reaction, and that is the tropical disease frambesia, or yaws, caused by a spirochete almost indistinguishable from the spirochete of syphilis. However, there are numerous technical sources of error in the conduct of Wassermann reaction, and the technic is a matter of primary importance; but we have gained a great deal of new knowledge on this subject. I believe we should make our Wassermann reaction just as sensitive as is possible for maintaining its specificity for syphilis, and after the primary stage of the disease I believe that the Wassermann reaction becomes one of the most reliable single symptoms of this disease.

Now the question arises, should or does a positive Wassermann reaction indicate always the presence of living spirochetes in the body? Should treatment be persisted in until the Wassermann reaction is eradicated and kept negative over a period of years?

We cannot answer these questions in a positive manner, but I think our knowledge indicates that a continuously positive Wassermann can be taken as an indication of the presence of living spirochetes in the tissues. However, positive Wassermann reaction is not always accompanied by evidence of syphilis, and in my opinion one possible reason is that the spirochetes are located in physiologically unimportant tissues, that is, tissues that may be the seat of chronic pathological changes without marked signs or symptoms of disease. Therefore, I believe the Wassermann reaction can be persistently positive in cases that do not present definite clinical evidence of this disease, particularly in the latent stages, but with the danger and likelihood of development of active lesions at any time. We know that it is not always possible to eradicate the Wassermann reaction when a sensitive technic is employed, but I believe we should treat these "Wassermann-fast" cases intermittently and keep them more or less under direct supervision.

When the Wassermann reaction is used as a serological guide to treatment, it should be as sensitive as we can make it and maintain its specificity. The Wassermann reaction is not too sensitive, rather it is not sensitive enough. The great biological error is the fact that it fails to detect all cases of syphilis. We know we must have a minimal degree of spirochetal activity before we may detect in the test-tube the reagin or so-called antibody producing this reaction. Therefore, we should not lay too much emphasis upon a negative reaction, and particularly if the technic is known to lack in sensitiveness, as when syphilitic liver antigen is employed. In conducting the Wassermann reaction for the serological guide to the treatment of syphilis, I think it is well for the patient's serum to be used in graduated amounts, using a single amount of serum, as is the general custom. Not infrequently the physician receives a report of ++++ repeatedly at intervals in spite of active treatment and concludes that the treatment administered has had no influence upon the disease, whereas if the serologist uses graduated amounts of serum in conducting the test, evidences of improvement could be brought out which are alike encouraging to the physician and patient.

According to present opinion, the Wassermann reaction should be negative consecutively over a period of at least one and possibly two years before cure may be said to have been effected. No physician should make the mistake of discharging his patient from treatment on the basis of a single negative reaction, and, furthermore, after a series of negative Wassermanns on the blood the spinal fluid should be examined, because in some cases the Wassermann is positive in spinal fluid when negative in the blood. This is probably due, in part at least, to the fact that spinal fluid is tested in amounts five to ten times larger than is permissible with serums, which increases the sensitiveness of the reaction with spinal fluid.

Dr. Jay F. Schamberg: The measure of value of any remedy or drug in the treatment of syphilis will depend upon the relationship of its chemical affinity for the spirochete as compared with its affinity for the body cells. No active remedy which destroys the spirochete can fail to have some influence upon the cells of the body. We know that the arsphenamins, among other influences, exert their dominant toxic influence upon the liver; therefore, the caution which Dr. McCrae and Dr. Stevens have given us this evening to avoid these drugs in the presence of a hepatitis. Mercury, on the other hand, exerts its dominant toxic effect upon the kidneys. The obvious aim of treatment in syphilis is to sterilize the patient of all the invading parasites, but this must be accomplished with due consideration for the maintenance of the physical integrity of the patient's organs. In our desire to cure the patient we must not abbreviate his life with the vigor of the treatment we carry out. The vigor of treatment in syphilis will depend upon the age of the patient, upon his general strength and upon the age of the infection. I cannot too strongly commend the wise words expressed here this evening by Dr. McCrae concerning the treatment of syphilis in differentiating the treatment of recent syphilis and of chronic syphilis. In recent syphilis our goal is to exterminate all the spirochetes in the body and thus effect a cure. In chronic syphilis, where changes have taken place in the tissues and organs, our goal is to dissipate the existing clinical symptoms and give such systematic treatment as will tend to arrest the progress of the disease. I am afraid in the past we have prosecuted the extinction of the Wassermann reaction too much as a fetich. There has been too intense a desire on our part to efface or reverse the Wassermann reaction. The Wassermann reaction, as I take it, is a measure of spirochetal activity in the body at the time the test is taken. But its effacement or reversal should not be the end of all treatment. In patients of fairly advanced years with visceral or nerve syphilis a persistent positive Wassermann should be to us a matter of relative indifference. We have all seen such patients alive after many decades in reasonably good health. The real aim of treatment is the removal of syphilitic manifestations and a consistent and conscientious effort to arrest any further progress of the disease.

Now there is perhaps no detailed therapeutic program in the treatment of syphilis which would command the acquiescence of all clinicians. Each man of experience has his own pet methods, but there is one thing, I think, upon which all are agreed, that is, in recent or early syphilis there must be an irreducible minimum of treatment throughout the first and second years at any rate, in order to prevent recurrences, and this irrespective of the outcome of the Wassermann test. I am afraid that we physicians have allowed our patients to concentrate their attention too much upon

the Wassermann reaction. They are told that the reaction is one plus or three plus or four plus, and their mind is correspondingly elated or depressed according to the outcome. I think we should attempt to withhold this knowledge from the patient as far as possible and reserve the information for our own therapeutic guidance. There is no doubt, however, as to the enormous value of this test both for diagnosis and for treatment.

Time will not permit me to say more than a few words in regard to the remedies employed in syphilis. There are perhaps four groups—the arsphenamins, mercury, bismuth and the iodides. Of the arsphenamins I prefer neoarsphenamin in general, not by reason of its greater facility of administration but by reason of the better effect in the experimental animals. It gives a higher therapeutic index; furthermore, it is better tolerated by patients, thus lessening the liability to interruption of treatment by severe reactions and thus favoring systematic care of patients. Bismuth is a new drug in syphilis. It has distinct spirocheticide properties and is capable of effecting the disappearance of existing manifestations. While its value cannot be definitely stated at the present time, it is probably comparable with mercury. There is a new drug just being introduced—sulpharsphenamin—which, though therapeutically inferior to neoarsphenamin, has the advantage that it is better tolerated by intramuscular injection. It can be dissolved in a small amount of water and administered in the gluteal region. In no disease, perhaps, more than in syphilis is individualization in treatment necessary. No one can set forth a therapeutic program which would be acceptable to all. New remedies and new methods are being introduced so rapidly that, like the railroad schedule, the treatment is subject to change without notice.

THE PHYSIOLOGICAL FUNCTIONS OF INSULIN

The Mary Scott Newbold Lecture

Lecture X[1]

By Professor J. J. R. MacLEOD, M.D.
TORONTO, CANADA

I wish to say that I consider it a great honor, indeed, to be asked to address a society such as this that stands for the best in medical science.

In introducing the subject which I have been asked to talk to you about tonight, I would like to remind you that the earliest investigations showing that there is a relationship between the pancreas and diabetes extend as far back as the seventeenth century, when Conrad Brunner showed that the pancreas could be excised in dogs without any definitely unfavorable symptoms supervening. The experiments were done with the object of showing that this gland is not essential for digestion, but incidentally it was noted that the animals behaved in a most peculiar manner. They exhibited definite symptoms of hunger and of diuresis. It is also interesting to note that an even earlier investigator, Malpighi, as a result of what he described as excision of the spleen, noted also in dogs that the animals exhibited symptoms which seem to have been those of diabetes. The chief outcome of Brunner's investigations was to divert men's minds from the pancreas, for it seemed from his results, and those of his contemporary Peyer, to be definitely established that this gland could have no essential function in the well-being of the animal. Even in the text-book of Johannes

[1] Read March 7, 1923.

Müller, published during the first quarter of the nineteenth century, only a few paragraphs are devoted to the function of the pancreas, many pages, however, being devoted to the function of the stomach, for by that time the work of William Beaumont done in this country had become known in Europe. It was really from clinical sources that the incentive to further investigation of the pancreatic function came. Cowley and Bright, and later the French physician Bouchardat, showed that there is a definite relationship between disease of this gland and diabetes in man. The belief in this association gradually grew, grew in that peculiar way in which impressions do grow in every science, but particularly in medical science, and men's minds became more and more directed to on the possible relationship between the pancreatic function and the disease diabetes. Claude Bernard, among others, was attracted by this possibility, and although most of this master's work on the pancreas pertained to its digestive function, he also endeavored to seek for its possible relationship to diabetes. He tried to remove the antidiabetic function of the gland in various ways by injecting its ducts with oil or by ligating them, or by extirpating the gland, but he observed no symptoms like those of diabetes to become developed.

About 1869, Langerhans, a pupil of Virchow, described the islands now known by his name, and some years later Kühne and Lea described their interesting blood supply, and, in 1889, von Mering and Minkowski made the epoch-making discovery that complete excision of the pancreas is definitely followed by the characteristic symptoms of diabetes. This great discovery naturally attracted very widespread attention by numerous types of investigators—anatomists, experimentalists and clinicians. The anatomists such as Laguesse and Diamare contributed the most important work at this stage, and many experimentalists repeated Minkowski's observations and supplied important confirmation of the results. Minkowski had suggested in a paper published in 1892 that removal of the pancreas must result in the removal from the body of something that is necessary for the metabolism of carbohydrates, but he was rather vague as to what this something might be. The

statement that it was an internal secretion was first definitely made by Lepine, whose work you are probably more or less familiar with. That the pancreas produces an internal secretion having to do with carbohydrate metabolism and with the regulation of the blood sugar came to be generally recognized, particularly because about this time the conception of internal secretions had taken firm root through the discoveries of thyroid and of suprarenal extract. The anatomists pointed out that the structures in the pancreas that are probably responsible for this internal secretion of the gland are the islands of Langerhans.

Stimulated by the work of these investigators, many attempts were made to put the hypothesis to the crucial test, which in this case is obviously to administer an extract of the gland to animals rendered diabetic by its removal. The fact that the removal of a gland is followed by certain symptoms, as is the case with the pancreas and those of diabetes, is no proof that the gland produces an internal secretion, for it is obvious that the removal may cause the symptoms merely because the blood can no longer circulate through the gland where normally, some toxic substance is removed from it.

These early attempts at the administration of pancreatic extracts were more or less failures, and it was not until 1908 that a really successful extract of the pancreas was produced. In this year Zulzer, a German investigator, prepared an alcoholic extract that was capable of removing certain of the symptoms of diabetes in laboratory animals and also in man. He showed that this extract could, for example, antidote the hyperglycemia that is produced by epinephrin. Six clinical cases were also treated with Zulzer's extract. Several of them showed favorable results. Most of the symptoms of diabetes were temporarily removed, glycosuria and acetonuria particularly. The subjective condition of several of the patients was also definitely bettered, but unfortunately in other cases toxic symptoms supervened, so alarming in their nature that the treatment was abandoned. The subject seemed to be closed by unfavorable results published by Forschback from Minkowski's Clinic, and as a result clinical interest in pancreatic extracts almost entirely disappeared, and the possibility that an

internal pancreatic secretion might exist came to be the problem of the experimentalists. Among these were Knowlton and Starling, who about 1912 succeeded in showing that the addition of pancreatic extract to the fluid perfused through the isolated heart removed from diabetic animals is capable of increasing to some extent the power to utilize sugar. Such results were not obtained, in a sufficient number of experiments, however, to make them convincing.

The work then came to this country. Stimulated by Knowlton and Starling's reports, Murlin, Cremer, E. L. Scott and Kleiner took up the problem, each of them attempting to solve it by preparing extracts of the pancreas, by various methods, and studying their effects on diabetic (depancreated) animals. These investigators made the fatal mistake of observing only one symptom of diabetes at a time instead of observing two symptoms, and I believe that had they insisted on this criterion they would have been sufficiently convinced from their results that they really had demonstrated the hormone, that they would not have abandoned their researches.

The most important of all the work that was done in this country was probably that of Clark, then working under Professor W. G. McCallum in the pathological laboratory at Johns Hopkins. Clark made investigations on the effect of transfusing a saline solution through the pancreas and then through the heart. He found if he transfused Locke's solution containing sugar through the heart before sending it through the pancreas that the rate of sugar consumption was relatively slow. If, on the other hand, he transfused this solution first through the pancreas and then through the heart the rate of sugar consumption became markedly increased; an excellent demonstration, showing that by transfusing sugar through the pancreas something becomes added to it that activates the sugar-consuming power of the heart and makes it possible for more sugar to be used in a given time. Later on he made another discovery, namely, that although by transfusion through the pancreas alone, there is no change in the amount of sugar as estimated by the reduction methods, yet an alteration takes place in the rotary power of the sugar. This indicates that the sugar must become

changed in some way: must become in some way prepared for combustion. Evidently this has some bearing on the ultimate consumption that takes place in the heart, and it conforms with the more recent work of Hewitt and of Winter and Smith, which will be alluded to later on.

This leads us to the experiments which were done by Dr. Banting and Mr. Best in the physiological laboratories in Toronto. Since Dr. Banting will describe these experiments to you, I will pass on to review briefly some of the anatomic facts regarding the pancreas upon which the experiments of Dr. Banting and Mr. Best depended. It will save time if I do this mainly by means of lantern slides.

It has been known, as a result of the observations of many investigators, that ligation of the pancreatic duct when it is properly performed is followed very shortly by a degeneration of the pancreatic tissue involving first mainly the zymogenous cells. Later, however, the island tissue also becomes involved. At a certain stage both the zymogenous tissue and the island cells are in a state of degeneration, but very early these tissues begin to be replaced by new tissue. These processes of degeneration or destruction and of regeneration go on in both the zymogenous and the island cells for many weeks, at least in the case of the rabbit, in which very accurate observations have been made. Ultimately, however, it is the island tissue that alone remains. The zymogenous tissue gradually disappears until finally, in about 550 days, in the rabbit, there remains practically nothing but island tissue. (Clark and Bensley.) It is a most interesting thing that this regenerated island tissue is definitely related to the duct system, and that in place of being scattered in small nodules, as it were, through the pancreas it extends along the duct system just as Dr. Slater Jackson has found it to be in the more primitive pancreas of the skate. It is for that reason that I call your attention to the peculiar distribution of the islet tissue in the pancreas of the skate (slide). These facts raise the question as to possible regeneration of islet cells in the diseased gland. It is evident, I think, if a degenerated pancreas many, many weeks after ligation of the ducts is capable of regenerating fresh island tissue from the tubules that

the same thing is likely to happen when the gland has been destroyed by disease, provided this has not involved the gland so extensively that no healthy duct cells are left. Where the gland is completely destroyed so that its duct system, as well as its secreting cells, have been destroyed, then no hope of regeneration can be entertained. Where, however, this process has not yet caused complete destruction, then it seems reasonable to expect that if we can rest the gland, either by dietetic treatment or by the administration of insulin, there is a chance that regeneration may be possible.

Banting and Best took advantage of these facts to prepare an extract of pancreas that would not contain the proteolytic ferment, trypsin, by whose action, in the extracts of the intact gland, they assumed destruction of the hormone would occur. They found that this extract lowers the percentage of sugar in the blood and diminishes the amount of sugar in the urine in depancreated dogs. By extraction of the intact gland with alcohol they also found that the antidiabetic hormone was sufficiently preserved, and later J. B. Collip succeeded in isolating from such extracts by precipitation with alcohol in higher concentrations, a fraction which could be used for administration to patients without causing local irritation.

The investigation at this stage was greatly advanced by the discovery that the blood sugar of normal rabbits is also affected by injection of the purified extracts to which the name insulin now came to be given, a name previously suggested for the then hypothetical hormone by Sir E. Sharpy Schafer.

(Here charts were shown illustrating the effect on the blood sugar of normal rabbits following the injection of extracts.)

It was also observed that the injected animals developed symptoms (convulsions) and that these symptoms appeared almost invariably when the blood sugar had fallen to somewhere about 40 or 45 mg. per cent. Occasionally so definite a parallelism between the convulsions and the percentage of blood sugar was not observed. Sometimes, for example, the convulsions appeared when the blood-sugar reading was only 60 mg. or they might fail

to appear, though it was considerably below 40 mg. If we take the average of a great many results we find, however, that convulsions appear almost invariably at about 45 mg. of sugar. These convulsions are quite characteristic. The animal shows clonic convulsions of its musculature which occasionally involves the whole of the body, and during this condition the animal with its head retracted throws itself about often in a very violent fashion exactly in the same way as occurs when an animal has been spun around and thrown on the floor. This would seem to indicate that there must be violent stimulation of the labyrinthine apparatus, the apparatus responsible for the control of equilibrium. The convulsions usually last for the best part of a minute, sometimes longer, and then the animal lies in a collapsed state, sometimes unconscious, breathing rapidly, the head usually retracted. It may lie in this condition for several minutes and then a second convulsion supervenes. These convulsive seizures alternating with periods of coma, may go on for several hours before the animal dies. It eventually dies of respiratory failure. The animal often becomes highly excitable before the actual convulsive seizures appear. The association between the incidence of these symptoms and the percentage of blood sugar indicates that the two must be correlated. The symptoms must be related to a lack of blood sugar. To confirm this we studied the effect on the symptoms of the injection of glucose. We found that the symptoms were immediately removed. There is nothing more striking that I know of in the physiological laboratory than to see the removal of those violent symptoms within a minute or so after the subcutaneous injection of glucose. No other blood sugar has anything like so satisfactory an effect. Levulose, mannose and galactose may diminish the symptoms, but they have no such definite an inhibiting effect as glucose. None of the dissaccharides injected subcutaneously or intravenously has any effect at all. Alkali has no effect. Nothing that we have tried save glucose definitely removes the symptoms except epinephrin, which, of course, mobilizes glucose in the body. This definitely shows that the symptoms are related to the lowering of the percentage of glucose in the blood.

It does not necessarily mean that they are directly due to the lowering of glucose; it merely shows they are closely related to it. Possibly something toxic develops in the body, particularly in the nerve cells, the development of which in the normal animal is prevented by a certain concentration of glucose or one of its close derivatives in the nerve cells; but that is a matter which needs further investigation. One cannot decide that by examining the blood. It is an intracellular process, not one that will involve the hydrogen-ion concentration of the blood. In this connection it is significant that injections of alkali do not relieve the symptoms.

Having demonstrated these facts for normal rabbits, the next thing was to find whether insulin is capable of preventing the various forms of hyperglycemia that are known to physiologists. Claude Bernard was the first to show that puncture of the floor of the fourth ventricle has the effect of causing marked glycosuria, but we found that insulin almost entirely prevents it, as well as the hyperglycemia which is its immediate cause. Various investigators, Stewart, Rogoff and others, have shown that asphyxia always causes a very marked hyperglycemia, but we have found that it fails to do so in animals injected with insulin. One form of asphyxial hyperglycemia that is quite common is that due to poisoning by carbon monoxide. Carbon monoxide in our experience, however, does not produce hyperglycemia after insulin. It may cause a slight rise, but this is very trivial. Ether also fails to cause the usual degree of hyperglycemia when it is given to rabbits injected with insulin. This is interesting in connection with surgical operations on diabetic patients since it must diminish the dangers due to hyperglycemia which are more serious in these than in normal patients. It is of practical importance to know that insulin will hold down ether hyperglycemia and thus diminish the risk. Another type of hyperglycemia that is well known to experimentalists is due to the subcutaneous injection of epinephrin. Now in this particular case it is clear that since you can grade the dose of epinephrin, and also the amount of insulin that is capable of preventing it, that it might be possible to determine the dosage of insulin in terms of the extent to which it can prevent the hyperglycemia due to epinephrin.

I want to say a few words about another part of our investigation, namely, a more comprehensive study of the effect of insulin on diabetic dogs. We studied these in greater detail than was possible at the time Banting and Best conducted their original experiments. The symptoms I wish to call your attention to are, first, the respiratory quotient; second, the glycogen metabolism, and third, fat metabolism. These were the three problems that Banting and Best could not investigate while they were at the main problem of ascertaining whether or not it was possible to isolate a pancreatic hormone. In regard to the respiratory quotient, you will remember that this expresses the relationship between the amount of carbon dioxide exhaled and the oxygen inhaled in a unit of time. It is a ratio which tells us what kind of food is undergoing metabolism. When carbohydrates are undergoing combustion the respiratory quotient is unity; there is as much carbon dioxide given out as of oxygen taken in, for the reason that carbohydrate consists essentially of carbon *plus* hydrogen and oxygen, in the same proportion as in water (CH_2O). From the combustion point of view, therefore, we may regard the molecule as being just so much carbon. When C is burned by O_2 the volume of O_2 that is used is equal to the volume of the CO_2 produced; therefore the respiratory quotient must be unity. It is different with fat and protein. In the case of fat and protein oxygen is required, not only for the carbon but for part of the hydrogen as well; therefore, the volume of oxygen which is used will be greater than the volume of carbon dioxide which is produced. The respiratory quotient, therefore, is less than unity, and it is one of the achievements of modern medical science, due to the work of Rubner, Lusk, Benedict and others, that it is now possible to tell precisely from the respiratory quotient what type of combustion is going on in the body at a given time, what portion of protein, what portion of carbohydrate is being burned. One may define diabetes as the inability of the body to burn carbohydrate. This means that the administration of carbohydrate to a diabetic patient will not cause the respiratory quotient to rise to the same extent as in a normal animal. There is no alteration of the respiratory quotient when diabetes is extreme,

such as in the case of diabetic dogs. If you feed diabetic dogs with carbohydrate, for example, there is little or no change in the respiratory quotient. If insulin be given along with sugar to a diabetic dog or patient, however, the respiratory quotient rises as in a normal individual. That then is a fundamental observation showing that insulin makes it possible for the sugar to be burned up. Now this is the last link in the chain of metabolic processes through which carbohydrate passes in the animal body, and the first link, as you all know, is the deposition of glycogen. In diabetes, both in laboratory animals and in man, no glycogen, or only a trace, is deposited in the liver, no matter how much sugar you may feed to the animal. If you give insulin along with sugar, however, glycogen becomes very abundant indeed in the liver. In some experiments on diabetic dogs we have observed over 12 per cent of glycogen in the livers, and in one case over 20 per cent was found. This is the highest percentage of glycogen that had been observed in any animal up to this time. This shows that insulin acts on the glycogenic end of the metabolic chain of carbohydrate as well as on its oxidative end. How these two processes are linked together we do not know, but that the two are affected together are obviously most significant facts.

With regard to fat metabolism, it is well known that in diabetes the process goes seriously wrong. In the first place the transportation of fat is affected so that the blood and the liver become loaded with this substance. The resulting lipemia is a serious symptom in diabetes, but it very quickly disappears on the administration of insulin. In the second place the combustion of fat which is at the other end of the metabolic chain is seriously deranged in diabetes. In a normal animal the combustion of fat proceeds by stages. The long fatty acid chain is broken off, bit by bit, by oxidation at the so-called beta C atom, and the end-products of this combustion are water and carbon dioxide. There is a stage in the breaking-down process, however, at which it is apt to hesitate, that is, at the stage of butyric acid ($CH_3CH_2CH_2COOH$), which it will be seen contains four carbon atoms. In diabetes butyric acid instead of becoming completely oxidized becomes so only as far as beta

oxybutyric acid ($CH_3CHOH\ CH_2COOH$) and the closely related acetoacetic acid and acetone, which are responsible for the symptoms of coma. It is said that this hesitation in the final oxidation of fatty acid is due to the fact that there is no carbohydrate combustion going on. Fat burns in the fire of carbohydrates, and since in the diabetic no carbohydrate is being burned, fat combustion also becomes incomplete. It smokes, as it were, and the smoke accumulates in the body in the form of beta oxybutyric acid. Since insulin affects the combustion of carbohydrates, it should also affect this ultimate combustion of fat, and such has been found to be the case. Insulin stirs up the carbohydrate fires so that fat combustion proceeds to its completion and the so-called acetone bodies disappear from the urine.

Finally, let me very, very briefly allude to some work I am engaged in pertaining to the mechanism of the action of insulin, but which is as yet incomplete. When rabbits are injected with varying quantities of insulin it is remarkable how during the first half-hour after injection the blood sugar always comes down at very nearly the same rate. This would seem to indicate that the process by which the blood sugar becomes lowered must be an intravascular one. It looks as if it must be something occurring in the blood itself. There are two reactions that might occur in blood itself. One is glycolysis and the other is a union of sugar with something else, or its polymerization into glycogen. If blood be removed from the body and insulin added to it, the rate of glycolysis is not altered. Neither is the rate of glycolysis different in the blood of a normal rabbit and in that of one previously injected with insulin. There is therefore no evidence that insulin influences the rate of glycolysis. The evidence for union of the glucose is also lacking, for if blood be taken from an animal when the hypoglycemia is marked and hydrolyzed by heating with acid there is no greater increase in reducing power than occurs in normal blood.

The conclusion is that insulin passes into the tissues, where it creates, as it were, a vacuum for sugar, with the result that sugar is withdrawn from the blood more quickly than the loss can be made

good by the mobilization of sugar from the glycogen of the liver. There is a sudden drainage of the sugar from the blood to fill up the vacuum for sugar in the tissues. That narrows the question down to this: What is the cause of the sugar vacuum in the tissues? There are several possibilities. One is that the sugar is oxidized; another is that it is reduced into a fat-like substance; and a third that it is converted into glycogen. With regard to oxidation we have seen that the respiratory quotient is very quickly raised when insulin is given to diabetic animals, indicating that the combustion of sugar is encouraged. That does not necessarily mean, however, that insulin would have the same effect in normal animals. This had to be specially investigated, which has been done by Mr. Pember and Mr. Dixon, who have demonstrated quite clearly that the respiratory quotient in normal animals is definitely raised by giving them insulin without any sugar. Not only that, but the energy output under these conditions becomes very greatly increased, at least in dogs. The behavior of the respiratory quotient in many of these experiments was remarkable, for it rose to well over unity and remained there for an hour or two. This may be due to a reduction of carbohydrate to fat, such as occurs, for example, in hibernating animals who eat great quantities of carbohydrate-rich food in the fall months and store it away as fat, during which time their respiratory quotient is well over unity. These respiratory experiments, therefore, indicate that there is increased combustion and that there is a change in the nature of metabolism. With regard to the third possibility, namely, the deposition of glycogen, the fact that insulin causes this to occur in the diabetic dog does not prove that it will also occur in the normal animal. As a matter of fact, we can obtain no evidence that it does, although we have paid much attention to the problem.

Regarding methods for the assay of insulin I may say that we are trying very hard to work this out on a satisfactory basis before it comes to the time when insulin will be available to everyone who wants to use it in the treatment of diabetes. At present we define as one physiological unit the amount that will lower the blood sugar of rabbits, starved for twenty-four hours, to the convulsive level (0.045 per cent) within three or four hours. The clinical

unit is one-third of this strength, because it has been found that some clinical cases benefit by one-third the physiological unit, and it is thought advisable to avoid fractions in dosage. It is difficult using normal rabbits, however, to obtain very accurate results, and Dr. Eadie has been trying to work out a more precise method by seeing to what extent insulin will prevent the hyperglycemia due to injections of glucose or injections of epinephrin.

Finally, let me allude to some experiments by Miss O'Brien and Mr. McCormick bearing on the possibility that the internal secretion (insulin) of the islands of Langerhans is controlled through the vagus nerve. You are aware that stimulation of the splanchnic nerve causes an increase in blood sugar, and the question arises whether the vagus by increasing the output of insulin might cause a lowering of blood sugar. Although many of the results are negative in character, there are some of them that clearly show a lowering of blood sugar following stimulation of the vagus.

With regard to the fundamental cause for the action of insulin, what does it do? Why does it make glucose be used up? The answer may be furnished by the experiments of Hewitt and Pryde, and more recently of Winter and Smith in England. The former observers have shown that ordinary glucose is converted into another form of glucose (gamma) when it comes in contact with living tissue cells. Ordinary glucose is called alpha-beta glucose and has a specific rotatory power which remains constant and corresponds to the reducing power. When this alpha-beta glucose comes in contact with insulin and certain tissue constituents some gamma glucose is formed, as is indicated by a lowering of the dextrorotatory process. Clark also found by transfusing Locke's solution containing glucose through the pancreas that although the reducing power was not altered its polarizing power became slightly lowered. It is very interesting that Winter and Smith have recently announced in the *British Medical Journal* that insulin is a substance which activates an enzyme derived from the liver to produce gamma glucose; that is, the combination of alpha and beta glucose *plus* liver extract *plus* insulin causes a production of gamma glucose. What the ultimate outcome of this experiment of Winter and Smith may be is very difficult to say.

THE PHYSIOLOGY OF THE LYMPHATICS

The Mary Scott Newbold Lecture

Lecture XI[1]

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The opportunity to deliver a Newbold Lecture is a highly prized distinction, which, I can assure you, is greatly appreciated by me as a guest of your country and your Society. And I feel this an obligation not easily to be fulfilled before this illustrious audience of the College of Physicians of Philadelphia, an institution which, together with the University of Pennsylvania, is universally acknowledged to have helped so materially toward the development of medical science both in the United States and in the world at large. The venerable portraits adorning this hall give historic evidence to this my contention.

The subject of my lecture is one which I hope may be thought worthy of the attention of this audience, as it treats of problems and questions which confront medical practice from several points of view and which have given occasion for highly suggestive research work, namely, the physiology of the lymphatics. The study of the physiology of the lymphatics or, as I should prefer to call it for the moment, the study of the formation of lymph, is undertaken not only for its own sake, but also as a part of a problem of far wider scope, the problem of permeability.

[1] Read April 4, 1923.

Let us begin with a short sketch of the morphological arrangement of the lymphatic system, because from this we shall be able to deduce some opinions to guide us to a physiological understanding of the physiology of the lymphatic system. As soon as the stage of multicellular organization is reached, we observe two systems of channels traversing the various tissues, the bloodvessels and the lymphatic vessels. Which of the two is developmentally the more recent has not yet been decided, and therefore we may leave that question out of our consideration. The smallest lymphatic vessels begin as interstices nearly everywhere between the cells of the body, so that in certain tissues good injection preparations of the lymphatic vessels give us the same appearance as if we had the whole organ before our eyes, so densely is the network of lymphatic vessels laid out. Of all structures the glandular ones are those which are richest in possession of lymphatic vessels, a fact of considerable physiological importance.

The very first beginnings of the lymphatic paths are still a matter of controversy. According to some anatomists of authority, they nowhere begin as open spaces between the tissue cells, but always are closed at their origin by a membrane; whereas according to other anatomists the finest subdivisions are open tissue spaces. Difficulties of technic lie at the bottom of this controversy, which for the sake of a clearer physiological insight, it is highly desirable to have settled definitely. From the standpoint of physiology it makes a great difference, whether the first beginning of the lymphatics is closed or open. If it is closed, then we have no less than three systems in the tissues, the blood, the lymphatics proper and interposed between capillaries and lymphatics a system which is called the tissue fluid. If we make this distinction, the consequence is, that we probably have to deal with a greater variety of processes of interchange of water and dissolved substances than is usually thought of, the interchange between blood capillaries and tissue fluid system, the interchange between the tissue fluid spaces and the various specific tissue cells, and, lastly, the interchange between tissue cells and lymphatic vessels proper.

The various lymphatic vessels all end in one or two major lymphatic ducts, which bring back the fluid derived from the various structures of the body to a venous bloodvessel not far from the right auricle of the heart. But if we follow the paths of the lymphatic vessels before they ultimately join the blood system, we meet with a fact which is highly suggestive. We find interposed structures, which we call lymph nodes or lymphatic glands and nowhere in the body is there any possibility of lymph entering the circulation before it has passed through lymphatic glands. Certainly this must have a meaning and I believe we are warranted in making the supposition, that evidently the lymph must undergo some transformation before it can be allowed to enter into the blood stream. We can put this supposition to an experimental test. Barbèra and myself, many years ago, expressed the view that the lymph coming from the tissues might carry with it waste products harmful to the organism, either absolutely or harmful only if they entered the circulation in too large a concentration. We artificially accelerated the lymph flow coming from the large duct, in which all the lymph is collected from the region of the head and neck of the left side. As a biological test of this fluid we used the reaction of the sensitive vasomotor centers of the medulla oblongata. We were able to show that the injection of an accelerated lymph (artificially hastened through the lymphatic glands) resulted in the appearance of the so-called Traube-Hering waves in the blood-pressure curve, a phenomenon known to arise when disturbances of the centers of the medulla occur. In properly conducted experiments there was never any sign of Traube-Hering waves or of some other changes which I am not going to dwell on, after injection either of arterial or of venous plasma. On the basis of this experiment we assumed, as a physiological function of the lymphatic glands, the ability to detoxicate certain products of tissue activity. Against these harmful products, under physiological conditions, the capillaries in the region where they originate are protected, since such products cannot enter the blood circulation, but must remain in the lymphatic vessels. Without a mechanism of the kind which we believe we have demonstrated in the

lymphatic glands, there would, of course, be no real safeguard. The function, which I am just ascribing to the lymphatic glands, is well known to pathologists. The lymphatic glands have always been taken as valuable protective structures against mechanical and chemical invaders of the organism. But we may vindicate this useful property of the lymphatic glands, also toward chemical substances, taking their origin in the body under physiological conditions. From our point of view it seems possible that certain diseases may have their origin in a deficiency of the lymphatic glands and that the popular view of the pathological significance of the lymphatic constitution has its foundation in physiological facts.

Just a word about this protection of the capillaries against the entry of harmful substances. A very instructive illustration of this protection is seen in the liver. The bile acids are very dangerous poisons and therefore cannot be permitted to enter the circulation. Under normal conditions they always find their way to the biliary ducts and neither into the blood nor into the lymph. To argue that this is a property of all secretions would not be correct; for instance, the enzymes secreted by the digestive glands can be detected in the blood. Even under unfavorable conditions, when the path for the bile acids toward the ducts is occluded, we see that these substances do not enter the capillary vessels in the liver, but that they leave the liver (as Fleischl and Vaughan Harley showed in Ludwig's institute) by way of the lymphatics. Although some observers do not agree with these observations, it is not difficult to verify them. We may generalize and say, that the capillaries are safeguarded against the entry of certain substances. If this is so, we must postulate some system toward which these substances can be diverted, and reasoning in this manner, we see the necessity for the lymphatic system. Not alone this, however, but something further follows from my deductions. If the lymphatic system contains substances derived from tissue activity, not capable of immediate entry into the blood circulation, then the origin of lymph might or must bear some relation to tissue activity. Here for the first time we come across a new

principle in the formation of lymph. We shall return to this principle later on. Historically the views as to lymph formation went in quite another direction. To get acquainted with and to discuss the various theories of lymph formation is not alone highly attractive, because we thereby learn something of the development of physiological ideas and experiments through modern masters of physiology, but it has also a practical importance, since these theories up to the present day have guided us in seeking an explanation for the facts observed.

It was Carl Ludwig who gave us the first real theory of lymph formation, a mechanical theory, according to which the formation of lymph was due to and dependent upon the difference of pressures inside and outside the capillaries. It is no mere chance, that just from Ludwig should have emanated this first purely mechanical theory. In the beginning of his great career he created methods of investigating blood-pressure and its variations, and in classical researches he laid the foundations of our present conceptions of hemodynamics. No doubt a man, who opens successfully quite new fields, must for psychological reasons, be biased in this direction. The more so, if the experiments allow an interpretation on lines which have become familiar to the investigator. The theory that the formation of lymph was a process of filtration, was chiefly based on experiments where pressure had been raised by venous obstruction. Certainly under this condition more lymph was formed and the arterial pressure, which was the one measured in these fundamental experiments, was augmented. Ludwig was perfectly justified in his conclusion, that blood-pressure, which was maintained and regulated by so many wonderful mechanisms, discovered by his own researches, was the dominating factor. The conception of permeability had in those days not yet been awarded much attention: Now we no longer maintain that venous obstruction affects only the capillary pressure, but recognize that it also changes the permeability of the capillary walls. There were other experiments adduced, in which there was a certain parallelism between the height of arterial pressure and the amount of lymph formation. But already, in these early times,

the far-reaching ideas of Ernst Heinrich Weber, which were only much later recognized in all their importance by Bayliss and Starling, might have precluded the conclusions drawn from mere measurements of arterial blood-pressure. Ludwig's theory nevertheless held the field, at least among physiologists, until the year 1891. Only the great creator of the mechanical theory, Ludwig himself, had more or less given it up, a fact, curiously enough, not generally known, although when one carefully reads the papers from his institution between the years 1866–1876, papers unequalled in the experimental treatment of the question of lymph formation, one scarcely finds in them a dogmatic instance on the filtration theory.

Heidenhain's classical paper on lymph formation of the year 1891 marks the next great step in the question of lymph formation. The advance in physiological science is evidenced very distinctly in this paper by the fact that, almost for the first time, the new ideas of physical chemistry are applied with all their force in the problems discussed. From the standpoint of physiology it is interesting to note that Heidenhain attacked the problem of lymph formation, because he was stimulated by two biological reasons. The one was, that he did not believe that the mechanism of filtration could produce lymph or tissue fluid, which would satisfy the various requirements of the tissues. He based his argument on the amount of calcium which is daily secreted by the milk gland of a cow. We know the amount of calcium present in the tissue fluid, we are able approximately to estimate the quantity of lymph formed; these two values never give the amount of calcium, which really is secreted by the milk gland and which primarily must have been delivered to the gland-cells from the blood-capillaries by way of the tissue-fluid. As filtration cannot account for the actual amounts, some other mechanism must be postulated to meet the demand. The other reason was a fact well known to pathologists, the curious idiosyncrasy of certain individuals to various substances which cause urticaria, a disturbance which might be looked upon as a local increase of lymph formation in the skin. Heidenhain began his researches by performing various experiments,

very ingeniously devised, with the idea of demonstrating that there was no parallelism between height of blood-pressure and the amount of lymph formation. He was able to show, that in cases of very low arterial pressure there was a marked augmentation of lymph flow, while with a high arterial pressure there is no corresponding increase. Far more important than this negative side of his research, was the beautiful discovery of substances called by him lymphagogues of the first class. These substances he discovered in extracts of crab muscles, extracts of mussels, extracts of leeches, in various other extracts of animal origin and in peptone. After intravenous injection of small amounts of these substances, there occurred a greatly increased flow of lymph from the thoracic duct, without being accompanied by an appreciable change in arterial pressure—with the exception, that after injection of peptone even a great fall of arterial blood-pressure could be observed —and without any great change of the osmotic properties of the blood, as only small amounts of the said substances were necessary to produce the effect. On the other hand the character of the lymph had also changed: it was far more concentrated than normal lymph and it had more or less lost the property of clotting. All these discoveries were the outcome of a happy union of medical and physiological thinking, a union, which ought always to be upheld. Heidenhain inferred from these facts that the lymphagogues of the first class stimulated the cells of the capillary walls and that under the influence of this stimulation the living endothelial cells secreted into the various tissues the tissue fluid required by the demands of the individual tissues. And he correlated this secretory power of the endothelial cells with a selective permeability of the bloodvessel walls, varying from tissue to tissue. In this way arose the famous secretory theory of Heidenhain and it will be readily understood, why this theory was especially appreciated by pathologists. Heidenhain, himself, however, much as he was imbued by a biological spirit, was far from being onesided, but wherever the experimental results clearly allowed it, he tried to explain the formation of lymph without resorting to specific secretion. This spirit he showed clearly in the last part of his

remarkable paper, where he investigated the effects of an intravenous injection of concentrated crystalloid solutions, which produce an enormous flow of lymph. The careful analysis of his own observations and of the many facts, shown by Ludwig's school to occur after intravenous injection of concentrated crystalloid solutions, brought Heidenhain to the conclusion, that the effects of the lymphagogues of the second class, as he called them, could be explained on a purely physico-chemical basis. The concentrated crystalloids rapidly leave the capillaries and by osmosis, water enters the tissue spaces and the capillaries coming from the tissue cells. The amount of water, too large to be taken up by the bloodvessels, is the origin of the greatly increased lymph flow. I should like to insist upon one point of great importance. By his explanation Heidenhain was the first to demonstrate in an unmistakable way that the source of lymph, which we collect from a lymphatic duct, is or may be a double one, the blood and the tissues. If that is so, it follows of necessity, that a study of lymph flow is not identical with a study of permeability of bloodvessel walls. The processes in the beginnings of the lymphatic system are far more complicated than mere results of interchange between the capillaries and tissue spaces would be.

The Heidenhain secretory theory and his physico-chemical views rapidly gained ground and I believe that among pathologists there are still a good many adherents of the Heidenhain theory. Personally I believe, although my views differ from those of Heidenhain, that his theory is still well worth serious consideration. The subsequent development of the question of lymph formation took the same trend as the very similar problem of urine secretion; in both cases we have two well-founded theories, a mechanical and a secretory theory—or, as I should prefer to call it, a biological theory. It was in the interest of the purely mechanical theory that Bayliss and Starling undertook a series of splendid experiments on lymph formation. In the first place they took up the above-mentioned views of E. H. Weber and showed in a most convincing manner, that the mere measurement of arterial pressure gave no reliable information about the capil-

lary pressure, and that this could only be gained if arterial and venous pressure are measured at the same time. A careful investigation of most of the favorite methods of altering the hemodynamic conditions in the organism revealed, that although the arterial pressure might be low, nevertheless the capillary pressure might be raised by a disproportionate augmentation of a venous pressure. For instance in hydremic plethora induced either by infusion of large amounts of normal saline solution or by injection of concentrated crystalloid solutions, arterial pressure is not changed, but as the venous pressure measured in the vena cava and in the portal vein is greatly increased, the same holds good also for the capillary pressure. In some of Heidenhain's experiments, where he had lowered blood-pressure, measurements of venous pressure showed, that the capillary pressure had not been lowered. Therefore Bayliss and Starling explained all those cases, where Heidenhain had failed to find a parallelism between blood-pressure and lymph formation, by the conditions of capillary pressure which could be stated as being in accordance with the demands of the filtration theory.

Also the effects of intravenous injection of concentrated crystalloid solutions on lymph formation they believe to be explainable on the basis of the filtration theory. As already stated, one of the consequences is hydremic plethora, but this is also combined with increased capillary pressure. Therefore the suppositions for the filtration theory hold good and Bayliss and Starling discard Heidenhain's physico-chemical explanation. As to the lymphagogues of the first class, Bayliss and Starling convinced themselves that they at least cannot be explained by blood-pressure changes bringing filtration into play. They were able to make a remarkable and very important discovery, namely, that the whole augmentation of lymph flow after intravenous injection of the lymphagogues of the first class totally disappeared after ligation of the portal lymphatics. Therefore the localization of the whole action of these lymphagogues must be in the liver. We shall further see what conclusions can be drawn from this discovery. Bayliss and Starling restricted themselves to the opinion that the

liver capillaries were especially permeable and were made even more so by the action of the lymphagogues. This supposition of a greater permeability of the liver capillaries compared with those of other regions they believe to explain the fact, that the lymph derived from the liver is found more concentrated than lymph coming from other tissues.

The brilliant researches of Bayliss and Starling restored the purely mechanical theory in the eyes of many.

Some years later we took up the question of lymph formation in the Berne Physiological Institute in researches which were conducted with the help of my late friend, Dr. Barbèra of Messina, and my American friends, Dr. Gies of Columbia University, and the late Dr. Busch of Buffalo. The starting point of these researches was first a biological idea, suggested by the structural arrangement of the lymphatic system. I have dwelt in a former part of my lecture on this suggestion, namely, that the origin and distribution of the lymphatic system in the tissues and the fact that all lymph must pass for detoxication through lymphatic glands—all point toward a close connection between lymph formation and tissue activity. Secondly, in such glands which secrete only under the influence of the nervous system, we are able to delineate with approximate accuracy the true sequence of events. The first thing which happens is, that nervous impulses reach the specific gland-cells and there awaken their specific action. This is the primary event—everything else which happens thereafter is secondary. The idea which guided us, therefore, was that tissue activity lay at the bottom of lymph formation, and that under physiological conditions tissue activity is the primary cause of lymph formation. We can review briefly the experimental facts which favor this view: Barbèra and I were able to show that activity of the salivary gland caused increased lymph formation: while the mere vasomotor changes in the salivary gland (vasodilatation, raised capillary pressure and accelerated blood flow) proved in our and in earlier experiments of Heidenhain to be without any influence on lymph formation. The ideal possibility of clean-cut separation of these two phenomena, which the salivary

gland so well permits, we can scarcely hope to find realized in other tissues. We will see later that we shall once more be able to draw important conclusions on a much-debated question of principle by using the salivary gland as the most favorable site for investigation.

If tissue activity is considered to be the principal primary cause of lymph formation under physiological conditions—under pathological conditions other factors may play the more important role—then one ought to be able to show that this view holds good in other regions of the body as well, and especially holds good for Heidenhain's lymphagogues of the first class. To begin with these lymphagogues, since Bayliss and Starling have shown that their action is localized in the liver, we ought to be able to show that the substances belonging to this class have an influence on liver activity. Indeed Barbèra and I were able to demonstrate that peptone, which belongs to this class of substances, produces an increased flow of bile, and bile formation is certainly an indication of liver activity. There is another property in common for all lymphagogues of the first class, namely, the abolition or lowering of coagulability of blood and lymph. This is so characteristic that one can even predict the lymphagogue character of an unknown substance, if it shows this property. But how is the failure of the blood to coagulate related to liver activity? The relation becomes clear as soon as we recognize the fact, that the equilibrium of the colloids of the blood is in part maintained by the secretion into the blood from the liver of a substance called anti-thrombin. The view that Heidenhain's lymphagogues of the first class are in reality liver poisons, as I have called them, is apt to shed light on a situation frequently met in clinical medicine. Over and over again the observation is made, that urticaria is connected in some way with indigestion and with a diseased state of the liver. In another way we have tried to show that peptone, one of the typical liver poisons, has a specific influence on the liver. We could demonstrate, that intravenous injection of peptone markedly changes the histological appearance of the liver cells. Even an abundant oral administration of Witte's peptone can cause

alterations in the liver cells. Dr. Pletnew, of Moscow, demonstrated in the Berne Institute, that the limit of sugar assimilation was greatly lowered when a dog was fed with abundant amounts of peptone, and Dr. Tschannen completed the evidence by showing that feeding rats with peptone in a few days made their liver perfectly free from glycogen. More recently Witte's peptone has been shown to be possessed of a specific motor influence on muscular structures of the hepatic channels. Pick, of Vienna, has brought forward evidence that peptone causes obstruction of the hepatic vein and that in consequence thereof we have the fatal results of stasis in the most extensive venous territory in the body. It might appear from this new discovery that the shock poisons—peptone belongs to the shock poisons—act by raised capillary pressure. Even if we had this raised capillary pressure, which is doubtful in the case of peptone, we would not alone have this, but also increased permeability which is always concomitant with venous obstruction. But not without reason I have tried to draw attention to the evidence we have as to the liver cells themselves; to point out that even if this mechanism of the hepatic vein were to hold good under the much milder conditions where peptone, strawberry extract, etc., act as lymphagogues, it would scarcely explain those facts and still less would it explain the actions of the other lymphagogues which do not belong, like peptone, to the type of substance which we classify as shock poisons. But more conclusive than all this is the fact that we can positively show that purely physiological liver activities cause an increased formation of lymph. It was my late friend, Dr. Busch, of Buffalo, who demonstrated in the Berne Institute that causing the liver to form urea or stimulating the liver to increased activity by bile acids or by hemoglobin, even glycogen formation, was accompanied by augmentation of lymph flow derived from the liver. He was also able to show, that the activity of the pancreas induced by secretin was likewise accompanied by an augmentation of lymph flow. Now, therefore, we have given proof of a close connection between tissue activity and lymph formation under more or less physiological conditions for the salivary gland,

for the liver and for the pancreas. I ought to add, that Prof. Hamburger, of Groningen, has demonstrated with a beautiful technic that the activity of the salivary glands of the horse was accompanied with increased lymph formation. It is most pleasing also that from Starling's laboratory came corroborations of the connection between salivary gland activity and lymph formation and between liver activity and lymph formation in the liver.

These facts, of which I have tried to give a brief review, laid the ground-work for a theory of lymph formation which differs from Heidenhain's in dispensing with a secretory function of the capillary endothelial cells, and from Starling's revived Ludwig filtration theory in not acknowledging the fundamental importance of capillary pressure. I even go so far as to believe that changes of capillary pressure alone under physiological conditions do not cause an interchange between capillaries and tissue fluid. We have therefore once more to take into consideration those observations which seem to be strongly in favor of the filtration theory. Bayliss and Starling, as already mentioned, showed that the hydremic plethora induced by injection of concentrated crystalloid solution (Heidenhain's lymphagogues of the second class) was due to increased capillary pressure. I believe nevertheless that it is possible to show that the raised capillary pressure, certainly present in these experiments, is not the cause for increased lymph formation. My friend Dr. Gies, when working in the Berne Institute, has enriched our knowledge of lymph formation by his study of one of the most remarkable examples of increased lymph formation, namely, postmortem lymph formation. By postmortem lymph formation of course I do not mean the well-known possibility of getting a certain amount of lymph after the death of an animal by employing pumping movements on the abdomen and the thorax. By these movements we do not produce anything new, but only evacuate the fluid formed before death. The conditions in Dr. Gies' experiments were quite different and I believe that the story of his discovery is not without interest. Dr. Gies and myself were occupied with the influence of protoplasmic poisons on lymph formation, a problem stimulated by

our view that tissue activity lay at the bottom of lymph formation. We had selected quinin as a general protoplasmic poison and wished to ascertain, whether the lymph formation after intravenous injection of concentrated crystalloid solution could be altered through the action of quinin. In this way we wanted to find out whether there was not in the action of lymphagogues of the second class a superimposed physiological factor as well as a decided mechanical, or rather physico-chemical, factor. We had injected into a dog a concentrated solution of glucose and shortly after a big flow of lymph began to appear. Much to our disappointment the dog died at that moment, either from narcosis or from the effect of quinin or from both causes together. We had given up the experiment already, but to our greatest astonishment the lymph flow went on augmenting and we had a very big lymph flow, which we observed for over three hours. It was exactly the kind of lymph flow which one is accustomed to see after injection of a concentrated crystalloid solution. I should like to insist upon the fact, that we carefully abstained from even touching the animal to avoid the slightest possible chance of a mechanical pumping of lymph. In this way Dr. Gies' experiment, far from being a failure, shed quite a new light on the origin of the augmented lymph flow following the injection of concentrated crystalloid solutions. We believed that if several hours after death the kind of lymph flow described could occur, it could not be due to increased capillary pressure. So long after death one does not believe in an effective capillary pressure. Dr. Gies investigated the conditions from which the occurrence of the postmortal lymph flow depended. He found, that if he killed the animal immediately after having finished the intravenous injection of the concentrated glucose solution, the marked increase of lymph flow did not occur; but if he allowed a period of four to six minutes to intervene before killing the animal, then the augmented lymph flow invariably appeared. It is easily seen that these findings of Dr. Gies are perfectly in harmony with the original explanation given by Heidenhain. In four to six minutes the greater part of the injected crystalloids had left the bloodvessels and had drawn fluid from

the tissue cells, by osmosis, in such quantities that a part of it made its appearance as augmented lymph flow.

Dr. Gies' experiments seemed to be decisive, but Bainbridge pointed out in a paper coming from Starling's Institute that the venous pressure, measured in the portal vein and in the vena cava, remained high after injection of concentrated crystalloid solutions, so that even one hour after death the capillary pressure could be high. The matter had therefore to be taken up once more. Mrs. Cuttat-Galitzka arranged her experiments (done in the Berne Physiological Institute) in such a way, that capillary pressure was experimentally diminished, After having injected the concentrated crystalloid solution she drew a certain amount of blood from the animal and in this way managed to lower the venous pressure. Notwithstanding this lowering of venous and consequently of capillary pressure the same augmentation of lymph flow was observed as in the experiments of Dr. Gies. We therefore believe, that capillary pressure may be dismissed as the factor to which the lymph flow, caused by the lymphagogues of the second class, is due.

Another question arises, namely, whether, although the chief factor involved in the lymphagogue action of concentrated crystalloid solutions is a purely physico-chemical one, there is not even here a physiological factor in action, but overpowered by the influence of the physico-chemical factor. We must not forget that injection of a concentrated crystalloid solution is something very unphysiological and that the organism is not adapted to such a rough insult, which is mainly answered in the same passive way as any dead membrane would do. On the other hand, it is well worth remembering that every crystalloid may have a stimulating effect on tissue cells and then we have what we are seeking for, *i. e.*, physiological activity. The experimental way of investigating how far this theoretical reasoning might be correct, was to inject far smaller quantities of concentrated crystalloid solution than Heidenhain, Starling and we others had until then employed. The results of these experiments, when I injected small quantities of concentrated crystalloid solutions, were very

suggestive in the direction of a physiological factor. The increase of lymph flow was in the beginning scarcely perceptible. But gradually it rose and, what was more important, not alone the amount but also the concentration of the lymph became larger, and these changes lasted over a longer period of time. As this chain of events does not happen during observations of protracted lymph flow, the complication must be ascribed to the effects which the injection of small amounts of concentrated crystalloid solutions have on tissue activity. This is the physiological factor of the effect of the lymphagogues of the second class. Quite recently Meyer-Bisch, working on very different lines, has come to the same conclusions that we did, namely, that even small quantities of salts might have an action on the interchange of fluid between the tissue cells and their surroundings. His experiments started from observations on the influence of various proteins on the interchange of fluid in the human body. He convinced himself, that very much the same interchange could be produced also by such small amounts of crystalloids as we had formerly used in our animal experiments on lymph formation. The experiments of Meyer-Bisch, performed for therapeutic reasons in the Göttingen Medical Clinic, show that the physiological view of lymph formation is able to bear fruits in a domain of practical importance.

The study of lymph formation does not allow, as I have already pointed out, an immediate insight into the means which are active in producing the interchange of fluid between the blood and the tissue fluid. Certainly the immediate method of approach would be the safer one, and by this I mean the investigation of the changes produced in the blood when it passes a region of perfectly controlled conditions—changes which, under such conditions would occur in the blood, are apt to give a clear answer. The very simple and exact method of investigating changes in the concentration of the blood showing either augmentation or diminution was worked out many years ago in Ludwig's institution. The estimation of hemoglobin in arterial and venous blood gives us an unmistakable answer as to the changes in concentration of the plasma, as under physiological conditions hemoglobin does

not leave the capillary walls during an experiment. This method was employed in the laboratory of Hans Horst Meyer, when the question was taken up whether under conditions of raised blood-pressure there was a concentration of blood; under conditions of lowered blood-pressure a dilution. In the former case filtration of water or of a watery solution through the capillary walls would be indicated. To raise blood-pressure adrenalin was used and a very marked concentration of blood was observed. These experiments were and are still often quoted in favor of the filtration theory. I have already mentioned the grave objections against these experiments, and therefore need not repeat them. The influence which blood-pressure has on the interchange of fluid between capillaries and tissue fluid had to be examined under conditions where nothing else has changed but the mechanical factor under investigation. An ideal place for this examination we found to be the submaxillary gland of the dog or the cat. When I raised the blood-pressure in the head region by occluding the abdominal aorta for about two minutes, and collected the venous blood coming out of the carefully isolated vein of the submaxillary gland, under avoidance of a mechanical obstruction to the blood circulation in the gland, I found not the slighest difference in the hemoglobin concentration of the arterial and venous blood. This means that with raised arterial blood-pressure, and with nothing else but that, there is no filtration of fluid through the capillary walls. Let us sum up once more the conditions of this experiment: Raised capillary pressure, augmented circulation through the gland, no change of permeability, no increased tissue activity. It was easy to demonstrate, that when the true factor which really causes fluid interchange comes into play, then the time of the experiment is quite sufficient to demonstrate concentration of blood. It is only necessary to stimulate for one minute the chorda tympani and to collect the blood from the vein of the submaxillary gland. We have then the same circulatory mechanical changes as before, but superimposed upon these tissue activity. The hemoglobin content of the venous blood is then very much greater than that of the arterial blood collected at the same time. These experi-

ments show a close parallelism with those which were formerly reported on lymph formation in the salivary glands: Tissue activity produces lymph formation and tissue activity produces concentration of the blood. The latter is the necessary precursor of the former. Perhaps if the inability of raised capillary pressure to cause any increased fluid interchange between blood and tissues had been ascertained in the beginning, there would not have been so much predilection for the filtration theory.

I will just mention that vasodilation produced by stimulating the chorda, without tissue activity—a combination that can be produced by giving atropin—also is devoid of any influence on fluid exchange.

However convincing the experiments just reported may appear, they hold good, if one is very critical, only for the salivary gland. We might here be face to face with quite special conditions, which in this spot only prohibited blood-pressure from acting as a force for filtration. This might be in connection with the special position of the salivary glands, which only change from the state of nearly perfect rest into that of functional activity when nervous impulses reach them. One might assume that for this reason there are some arrangements that safeguard them from the influences of blood-pressure, these influences being here without any biological purpose. All this is of course a mere hypothesis, but nevertheless we though it worth while to put it to an experimental test. Dr. Tani therefore undertook some time ago in Berne a series of experiments, where again he mechanically raised blood-pressure in the upper regions of the body by pressure on the abdominal aorta. He collected venous blood from two regions, from the ear and from a side branch of the jugular vein. This he compared with the concentration of arterial blood collected at the same time and also of the venous blood when the blood-pressure had resumed its normal level. The result of these experiments was, that also in these two other regions there was not the slightest sign of fluid leaving the bloodvessels under the influences of raised blood-pressure. Therefore I believe that we are warranted in generalizing that filtration is not a force which is employed

under physiological conditions to attain the ends of the organism respecting the interchange of fluid between bloodvessels and tissues.

Although only experimental results can decide the questions which we are discussing, it is not unworthy of a biologist to speculate on the significance of the belief that filtration plays no part in physiological fluid interchange. It is usually true that the wants of the organism do not appertain to all of its parts at the same time nor in the same degree. Therefore a force acting quite universally, as blood-pressure does, would not constitute a mechanism especially well adapted to the requirements of the organism, nor would it work in that economic manner which so wonderfully adorns animal mechanisms. What is really wanted is a mechanism acting strictly locally and just at the time when the requirements occur. Evidently the principle of tissue activity as "causa movens" for lymph formation and interchange between the capillaries and the tissues is of greater adaptability than the filtration principle.

This principle of tissue or cellular activity does not mean, as sometimes is surmised, the introduction of an unknown vital force, it rather means that the complex processes constituting the various phenomena of life of the cells must be regarded as potent factors in every step of metabolism. Permeability is one of the primary steps. In acknowledging the complexity of tissue activity we are confronted with the task of analyzing it. In this respect much work has to be done, where we are as yet at the beginning. In undertaking this analysis we shall have to be carefully on the watch for the help that physico-chemistry and chemistry can give us. In reviewing the facts which are at present at our disposal, there are several which seem to be arguments in favor of the intervention of much-studied physico-chemical processes. Among these I should like to mention colloidal pressure, a force discovered many years ago by Karl Spiro. Anything lowering the colloidal pressure or water-retaining power of the colloids either in the cells or in the blood, will tend to make a transference of water from one spot to the other more easy. By making practical applications of this theoretical reasoning, Bayliss as well as Ellinger

have achieved results of a great value in therapeutics. This success is no absolute proof of the correctness of the theoretical views; in fact some authors have explained these results on quite another basis. Another physico-chemical factor, which seems to come to the foreground during tissue activity, is a momentary change in hydrogen-ion concentration and in the equilibrium of actions. Both these factors could very well change the conditions of permeability. One of the most brilliant proofs of changed permeability during activity is Embden's demonstration that phosphorus leaves the muscle fiber as soon as it passes into a state of activity. Such instances as I have just given might easily be multiplied, but I will abstain, and before ending my address will briefly draw the attention of my audience to one application of the physiological theory of lymph formation which seems to give testimony to its practical usefulness. Ellinger, in his remarkable study on the physiology and pathology of edema, points out that there are edemas neither of a renal nor of a cardiac origin, and that in accord herewith they are refractory to the usual cardiac and renal therapeutic measures. This fact suggested to him that there might be another cause for this type of edema and he believed that it might be explained on the basis of my physiological theory of lymph formation. He argued that there might be influences at work diminishing tissue activity and, if therefore tissue activity could be raised, that these edemas might be lessened. Now the best agent that we have to stimulate tissue activity is the hormone of the thyroid gland. Ellinger's line of argument proved quite correct; for the treatment of these patients with thyroid extract made their edema disappear in a marvellous way. This success also favored the view that edemas neither of renal nor of cardiac origin are due to thyroid deficiency. Ellinger did not rest content with this practical success, but put the theoretical basis to an experimental test. In a very convincing manner he showed in various ways that extirpation of the thyroid gland retarded and diminished the interchange of fluid between blood and tissues, and that permeability could be restored to its normal state when active thyroid was administered. I believe that the demonstration of

the great influence of thyroid gland on the phenomena of permeability is one of the best proofs that we have of the superiority of the claims for tissue activity over other factors of a mere mechanical kind which are thought to be active in governing permeability. But theories, however much they may be thought to be a pleasing embellishment of scientific work, are after all only of secondary importance. If I have been able to show that the study of lymph formation, from the point of view that has guided us in our investigations, has a stimulating influence on questions which interest medicine in general, I should feel, that in a slight way I had fulfilled, as a physiologist, my duty toward my distinguished audience.

PRESENTATION OF A PORTRAIT OF PROFESSOR WILLIAM GIBSON, ON BEHALF OF HIS GRANDDAUGHTER, MISS ANNE L. GIBSON[1]

By ASTLEY P. C. ASHHURST, M.D.

Born shortly after the close of the War of the Revolution, a surgeon of militia during the War of 1812, Dr. Gibson survived the War of the Rebellion by a number of years. During his life of eighty years he spent more than half of them in Philadelphia, and came to consider himself a Philadelphian. He was born in Baltimore, and he came to Philadelphia the winter of 1806–07 to attend the medical lectures at the University of Pennsylvania. There he heard Dr. Physick lecture on surgery. He went home to his boarding house, sat before the open fire and mused on the wonderful things he had heard. His landlady entered suddenly and asked what he was thinking about; he said, "I was thinking that some day I am going to have that man's place." She laughed at him, but at the dinner table she told this to all the other students in the house, and they laughed at him, and thereafter called him the successor of Dr. Physick.

Then he went to Edinburgh to study medicine and graduated there in 1809; from there he went to London and had the opportunity of seeing in London the wounded who returned from the battle of Corunna, in which the British claimed the victory over the French, although they lost their gallant general, Sir John Moore, whose funeral, I should say burial (for there was no funeral in the darkness of night), has been made forever memorable by the celebrated Ode of Wolfe.

[1] Verbal communication made April 4, 1923.

While in London, Gibson became a member of the household of Charles Bell, afterward Sir Charles Bell, and lived with him for several months; he came back to his home in Baltimore in 1810, married in 1811, had a large family, eventually married a second time, and had another family.

In the War of 1812, as I have said, he was a surgeon of militia. There were riots in Baltimore in those days and he stationed himself near at hand in case there might be need of surgery. He had the good fortune to see a man shot in the groin, whereupon he rushed out from his hiding place and put his finger in the wound and stopped the hemorrhage; every time he removed his finger the hemorrhage started afresh, so he kept his finger in the wound and had the patient conveyed to the man's home a short distance away. With his free hand he enlarged the wound and with the assistance of a friend got a flexible probe, armed with a ligature, passed it around the bleeding-point, tied the ligature, evacuated the clots and found he had tied the common iliac artery just below the bifurcation of the aorta. Next he put a ligature on two holes in the intestines, reduced the bowels, and then on wiping away the clots again he found the distal end of the common iliac bleeding, so soon had the collateral circulation become established. He tied the distal end of the common iliac artery in the same manner, brought the abdominal wound together with adhesive plaster and took care of the patient for fifteen days until he died. It is not surprising he died in those days. He died of secondary hemorrhage and peritonitis. An autopsy was held and showed that the proximal ligature was still intact—the distal had become detached. Of the ligatures on the intestines one had held, the other had come loose, but the perforation had been sealed by adhesions; nevertheless, the man had peritonitis. Dr. Gibson very sensibly pointed out that the important lesson to be learned was not that you could do such an operation in an emergency, but that it had been demonstrated for the first time in the world that collateral circulation could be maintained in the lower extremity after ligation of the common iliac artery! For he said that such an operation some day might be undertaken on a patient

with aneurysm, and that if the patient was in reasonable health one could hope to have him survive the operation.

In the War of 1812, Dr. Gibson acquired further fame by operating on General Winfield Scott, who had been wounded in the Battle of Lundy's Lane, near Niagara Falls, and whose wound refused to heal. Dr. Gibson removed the bullet from his leg and General Scott was soon restored to a long and useful life.

About this same time, also, Gibson was active in establishing the medical department of the University of Maryland, and became its first professor of surgery. This school soon became a rival of the University of Pennsylvania.

By the year 1818 the medical faculty of the University of Pennsylvania was in a bad way. Most of the faculty had been lost by death within a few years. Rush had died in 1813, Benjamin Smith Barton in 1815, Caspar Wistar in 1818, and Dorsey, the "amiable Dorsey," as my father called him, who succeeded Wistar as professor of anatomy, died in 1818, a few days after he gave his introductory lecture. Dorsey sat up late at night writing his lecture, and became so absorbed in its preparation that he failed to notice that his fire had gone out. He delivered the lecture the next day, but got "a congestive chill" (pneumonia?) that night, and died in a few days. That left the University with three empty chairs. Canvass was on for these chairs and acrimonious discussion arose. Some people in Philadelphia wanted Dr. Thomas Hewson to succeed to the chair of anatomy, but the University faculty did not want that. I do not know why. Also, there was a party here in Philadelphia that wanted Dr. Hartshorne as professor of surgery. The University was against having "the Quaker party" introduced. It was said if these elections were made, Chapman, Physick and Dewees would resign and establish a rival school. So the Trustees persuaded old Dr. Physick, then past fifty years of age (in those days this was old), who had been active as surgeon for twenty-seven years already, and had resigned five years previously from the Pennsylvania Hospital on account of uncertain health; and who, it was well known, had become so averse to doing any operation

that it became necessary to set a guard around the house where he was taken to operate, for fear that he would escape before doing the operation—they persuaded Dr. Physick, I say, to let himself be transferred to the chair of anatomy. They had asked Dr. Gibson to take the chair of anatomy, but he had declined. They persuaded Dr. John Redman Coxe to give up chemistry, which he had been teaching along with materia medica, and to confine himself to the latter. This left the chair of chemistry available for Dr. Robert Hare, even at that time one of the most distinguished chemists in the world. These arrangements left the chair of surgery vacant; and to it they elected Dr. Gibson, and he came to Philadelphia in the fall of 1819.

Perhaps there was yet another reason why it was desirable to fill the chair of anatomy by some respectable person like Physick. This was the contemplated arrival in Philadelphia about this time of that gadfly of medical politics, one Granville Sharp Pattison. This young Scotchman brought letters from distinguished physicians in Great Britain and was well received, but the chair of anatomy had been filled. It seems that he had been in correspondence with some physicians here who thought he would get the appointment if he were on the spot. But not long after his arrival, in the words used in after years by the editor of *The Lancet*, "his tarnished reputation" followed close on his heels. He left Glasgow soon after one of his colleagues, a Dr. Ure, had secured a divorce from his wife, having charged her with adultery with Mr. Pattison. Now, Mr. Pattison, being defeated in his ambition to become professor of anatomy in the University of Pennsylvania, established himself as a private teacher of anatomy and surgery. About the time he did this he published an article which was the first thing that brought him into controversy; he published an article entitled, "Experimental Observations on the Operation of Lithotomy," and in that article he claimed to have discovered a new fascia, "the prostate fascia," which nobody had ever heard of before. He had shown a dissection of this fascia to Dr. Physick, who said, "Very interesting." It may be remarked that while Physick was an excellent surgeon,

he was a very mediocre anatomist; and if he acknowledged he had never seen this fascia before, his qualifications as anatomist must be borne in mind. When Pattison's essay appeared, Gibson thought it incumbent on him to take up the defence of former anatomists and surgeons. The habit then was to prefix to one's pamphlets a quotation. This is the quotation Dr. Gibson prefixed to his: "Novelty captivates the superficial and thoughtless; vehemence delights the discontented and turbulent. He that contradicts acknowledged truth will always have an audience; he that vilifies established authority will always find abettors." Needless to say, these sonorous phrases were written by England's great lexicographer.

The objections urged by Gibson and other writers against Pattison's views were twofold: First, that the fascia he described was already well known, and second, that it was of no importance in the operation of lithotomy. And both these contentions hold true at the present day.

Whereupon, Pattison replied in another pamplet; he likened Dr. Gibson, sitting in the chair of surgery in the University, high and mighty, surrounded by a large family, rich and prosperous—he likened him to Haman in the Bible, and, quoting from the Book of Esther, he said, "Yet all this availeth me" (Haman) "nothing so long as I see Mordecai the Jew sitting in the King's Gate"—Mordecai the upright and just, representing Pattison himself. This was the way the controversy went on.

Now on top of all this, Dr. Nathaniel Chapman, who was professor of the institutes of medicine in the University, thought it was inimical to the University to have Mr. Pattison succeed so well in his private teaching; so he sent to Scotland and got a transcript of the testimony in this divorce trial, and published it, and above 8000 copies were circulated. So Mr. Pattison had to reply to this, and another pamphlet war was on. Meanwhile, Pattison found it too hot in Philadelphia and retired to Baltimore, where he took the position in the University of Maryland which Dr. Gibson had held. But Pattison did not remain there long. Mr. Wackley, the vituperative editor of *The Lancet*, said in later

WILLIAM GIBSON

1788–1868

years that Mr. Pattison, on account of the gay life he led in Baltimore, failed in health and had to retire. At any rate, he went back to England, there became professor of surgery in the newly instituted University of London, but was there only for a very short time. He went there in 1827 and in 1831 he was ejected, ostensibly because the students would not stand him (of course he was a Scotchman and nothing Scotch was popular in London): the students claimed his lectures were monotonous in form and antiquated in substance; and they mutinied; and so the Board of Governors ejected Mr. Pattison. He then came back to Philadelphia as professor of anatomy in the Jefferson Medical College, which had been newly instituted by Dr. McClellan; and Pattison had not been there very long before he took part in an acrimonious discussion on the surgery of the parotid gland; he supported McClellan in claiming it had been completely extirpated a number of times by McClellan and others, with success. Dr. Gibson denied that it could be done or ever had been done, and again they had controversial correspondence in pamphlets.

In the meantime, Dr. Gibson was teaching surgery very efficiently. He gave good, systematic lectures; he had a museum and apparatus and operated upon the cadaver before the students, which had never been done before apparently; had magnified drawings made, and enlarged wax models, so that not only those on the front row could see, but all. He wrote a text-book on surgery which went through eight editions, and from which most American surgeons secured their basic education in surgery during a period of thirty years. The first edition, published in 1824, was dedicated to Physick, and from the almost lavish praise which Gibson throughout his career bestowed upon Physick, it would appear there could not have been anything unpleasant between them.

Gibson was the first to introduce clinical teaching by means of the ward class, as we know it today. Instead of carrying a group of students through the wards and making more or less desultory remarks on the patients as they were encountered, he collected

groups of similar cases from the various wards of the Almshouse, brought them before the students in the amphitheatre, and thus was enabled to supply his students with systematic clinical instruction. His constant iteration and reiteration, that it is the principles which must be taught students first of all, is well exemplified in the fine portrait of him which hangs in a place of honor in one of the University's halls, where beside him on a column appear the words, "Principles, Principles, Principles."

Dr. Gibson added constantly to his reputation as surgeon and operator. Especially notable was his performance of the Cesarean operation twice successfully on the same patient, in 1835 and 1837, long before the days of asepsis and even many years before the advent of surgical anesthesia. This was a truly notable achievement. He devised the operation of linear extraction of cataract in 1811; he operated for strabismus in 1818, twenty-one years before Dieffenbach's work on the same subject; he invented a circular saw (run by a clock spring) for work on the bones; he was the first in America to do suprapubic lithotomy; and he had the honesty and the courage to report in detail two disastrous experiences in which he ruptured the axillary artery in trying to reduce old dislocations of the shoulder.

When nearly seventy years of age, Dr. Gibson resigned his professorship (1855) and went again to Europe, where he had been on various trips before. Then he determined that he would spend the year as he pleased, and he found that, apart from the island of Guernsey, no place suited him so well as Newport in the summer and Florida or Georgia in the winter. Eventually, at eighty years of age, when on a fishing trip in Georgia, he developed "mortification of the toe," probably senile gangrene, and he died from that.

Now Dr. Gibson, as old Dr. Gross has said, was not an amiable man. He was not popular in Philadelphia. He was rather conceited and vain. For instance, when he came from Europe, on one trip, he brought a large wardrobe of garments and appeared each day in a new outfit of remarkable checks and stripes, and thought the uproarious applause of the students intended for

him, not for his clothes. He was not a popular man socially. He met my grandfather on the street one day (my grandfather and old Dr. Gibson's son had married sisters, daughters of Manuel Eyre, the younger), and Dr. Gibson asked my grandfather how his wife was. My grandfather said she was not very well that day. "Huh!" said Dr. Gibson, "Overeaten herself, I suppose; the Eyres were always gluttons." So he was not very much liked. One of his granddaughters tells this story: Sitting at the foot of his table with a carving-knife and fork, one of his sons annoyed him and he told him to be quiet, and the boy did not stop; and then he told him again to be quiet, and the son kept on. Thereupon, Gibson thrust the carving-fork in his hand through the boy's foot into the floor, and said, "Now will you be quiet?" So you see he must have been rather an unpleasant man in some ways.

Dr. Gibson was never a Fellow of the College (for in his day this was a College of *Physicians*, not in name only but in spirit and in truth), but, nevertheless, I am glad we have come into possession of this picture. The portrait was painted in 1819 on wood, by an artist named Wood, and has, to my mind, a rather wooden expression of countenance. Joseph Wood, the artist, was well known at that time for his paintings of this "Kit-Kat" size and for his miniatures. Born in New York State about 1778, he came to Philadelphia in 1813, and is known to have remained here as late as 1817, going subsequently to Washington, D. C. He may have painted Dr. Gibson either in Baltimore or Philadelphia.

So, Mr. President, I am very happy to be able to present to the College, on behalf of his granddaughter, Miss Anne L. Gibson, this little picture of a great teacher and a great surgeon.

HEALTH WORK IN THE PUBLIC SCHOOLS[1]

By DR. EDWIN C. BROOME
SUPERINTENDENT OF PUBLIC SCHOOLS IN PHILADELPHIA

The health work in our public schools is an important branch of our educational enterprise. There are 74 doctors employed for three hours daily, 9 medical supervisors, 1 general director and 80 nurses on full time. All the pupils are examined at least once a year for the purpose of discovering physical defects. Also, the symptoms of contagious diseases are carefully sought out, and there is full coöperation with the Board of Health for the purpose of preventing the spread of contagion in the schools.

In addition to this work, the doctors make regular sanitary inspections of schools and report on the same.

The following table will indicate the progress which has been made in the work of medical inspection since 1920:

COMPARATIVE RESULTS OF MEDICAL INSPECTION IN SCHOOLS

	1920.	1922.	Gain per cent.
Pupils enrolled	230,740	250,169	8.0
Per cent examined	79	92.0	13.0
Defects discovered	178,598	248,360	39.0
Per cent corrected	45	48.6	3.6
Dental cases	66,010	91,969	39.0
Number of nurses' visits to homes	7,598	9,247	21.5
Number of children taken to dispensary by nurses	15,599	22,875	46.5
Number of pupils per doctor	3,846	3,381	13.5[2]
Number of pupils per nurse	5,769	3,127	84.5[2]

[1] Read May 2, 1923.
[2] Gain in this item means reduction in proportion of pupils to doctor or nurse.

In connection with the health work in the schools, I should mention the work of nutrition. There are 80 groups of children, known as "nutrition groups," with about 25 children in each group, or about 2000 in all. These children are taught at stated periods and are given a special milk diet. They are also regularly supervised by the school nurses. The parents of these children are brought together each week. They are shown the health charts of the children and receive instructions as to how to regulate in the home the matters of sleep, diet, clothing, exercise, etc. There is also milk service in 132 schools. Approximately 17,000 half-pint bottles of milk are consumed daily in the elementary schools. Records of the gains made by the children are kept, and the results show a notable gain in the general health of those who have this milk diet.

There are 8 open-window or nutrition classes, containing about 200 pupils. These pupils remain all day in these classes and are under the direction of a teacher especially trained for the work. There are also 7 open-air classes for tuberculous children. These classes are also under the instruction of a specially trained teacher. The children also receive special medical inspection and the special services of a school nurse. The Board of Education supplies matrons for the classes of tuberculous children.

In this connection we should also think of the orthopedic classes. There are 17 of these, including 375 pupils; also, there are two classes in hospitals and a class at Brown's Farms. In the case of these latter classes the Board of Education furnishes a teacher. There are 11 sight-saving classes, with a total enrolment of 150 pupils, and 5 classes for the deaf. These classes are placed in rooms which are especially adapted for the purpose, and, in the case of the sight-saving classes, the walls are treated and shades are provided in such a way as to favor the eye-sight of the children as much as possible.

The list of special classes would not be complete without mention of the "intubation" class for tracheotomy cases at the Philadelphia General Hospital. As in the case of the orthopedic classes in hospitals, the Board of Education provides a teacher for this

class, so that the little children who are compelled to remain in the hospital for several weeks or months may receive some instruction, that their education may not be neglected.

A legitimate part of health work in the schools is the safety work, which is conducted so generally throughout the elementary schools. In this connection I wish to make special mention of the safety patrols. These are squads of large boys who are organized to protect the small children, where schools are located on dangerous streets. There are 52 such patrols and 660 boys engaged in this work. I know of no city that has gone into this enterprise and has done it so well as Philadelphia is doing. Miss "Safety First," of the Philadelphia Rapid Transit Co., is responsible for the organization of the safety patrols. The records of trolley and automobile accidents show a marked decrease in minor accidents to children in the schools where the safety patrols are located. In all public schools, also, talks on safety are regularly given.

I could not conclude this paper without reference to the school playgrounds, as no school system can properly safeguard the health of its children for the present and for the future, if it does not provide adequate playground space and proper supervision for play. In Philadelphia there are 30 so-called "all-year" playgrounds. These all-year playgrounds are conducted on the schoolyards by teachers who have been especially trained as playground instructors. They are open after school hours and afford a fine opportunity for children to play in safety who otherwise would be running around the streets and back alleys. During the summer we conduct about 70 summer playgrounds. These are equipped with apparatus, seats for mothers, and are thoroughly and efficiently supervised. In our playground work there are 150 teachers engaged, 5 supervisors and 1 director. The average daily attendance of the all-year playgrounds is 3500, and of the summer playgrounds, 7000. This one criticism I should like to offer, and that is that Philadelphia has not yet made sufficient provision for play. While much progress has been made in recent years, we are still far short of the ideal in providing proper places and facilities for the children who will be the adults and citizens of the next generation.

DISCUSSION

Dr. Alexander C. Abbott: In looking over the figures on page 2 of Dr. Broome's outline of the work, I feel that while they represent a most gratifying showing for the period covered, yet to those who have been more or less concerned with the work since its beginning they tell only a small part of the story. As I look over the general outline on the first page and see the additions to medical inspection which have been made, I could not but think of the extraordinary growth this work has taken in the twenty years of its organization in Philadelphia. I am not sure who started it, but I know I was concerned with it from its very beginning. The main point is that when it was started we had a very much more limited program than has since developed. Before this work, our children in the public schools were getting no attention from anyone, teacher or doctor, and probably little at home. Medical inspection was then a pretty crude affair, but it is amazing how quickly the necessity for expansion was demonstrated and how quickly we realized that we were in a most comprehensive field of activity. In endeavoring to meet what we believe were legitimate demands, we encountered some very interesting objections. One of the first things was the number of children who had defective vision and needed attention and glasses. Many children had been sent to special schools because they could not keep up with their classes. On examination we found that the child was not mentally backward, but had such defective vision that it could not keep up with the class work. I recall one child who, after treatment and glasses, went to the window and on looking out said, "Oh! I never knew that it looked like that outside before." When we asked for glasses, we were told that this was carrying paternalism to the nth power, and what next would we want? If our laws demand that children go to school, then, I believe, it is our duty to see that these children are in physical condition to profit by going to school.

ACTIVITIES OF THE DEPARTMENT OF PUBLIC HEALTH[1]

(1920–1923)

By JAMES M. ANDERS, M.D.

The results of health activities are commonly measured by a comparison of vital statistics. As applied to Philadelphia, there has been a marked reduction in the mortality-rate during the last three years, the average rate being the lowest of any similar consecutive group of years in the history of the city. The average yearly death-rate for 1920–21–22 is 13.4 compared with 18.45 for the three years previous, 1917–18–19. The statistics of the latter period, however, were greatly influenced by the epidemic of influenza in 1918, while the death-rate of the last three years was affected to a great extent by the recurring epidemic during 1920.

A principal factor in the reduction of the general mortality has been a concentrated effort in the interest of infants, the death-rate of which has reached a lower level during the last three years than in any other like consecutive period. The average annual infant mortality-rate for 1920–21–22 was 82.8 per 1000 births as compared with 107.5 during 1917–18–19. Favorable records during 1922 prevail in spite of a lower birth-rate. The department has been influential in obtaining a more thorough registration of births in this city.

There has been a notable reduction in the death-rate of such principal diseases as typhoid fever, diphtheria, pulmonary tuberculosis, diarrhea, and enteritis in infants.

[1] Read May 2, 1923.

The results of the department's activities cannot be determined alone by the vital statistics during the last three years, since the benefits of the constructive program now nearly completed, will make themselves evident in the next few years.

A number of special activities have been conducted by the director's office, aided by the various divisions, and in some instances by civic organizations interested in health matters. During the summer of 1920 a vigorous rat campaign was carried out with a fair degree of success. In the spring of each year Public Health Day was observed in conjunction with medical and allied health and civic bodies.

Public Health Day has been celebrated in Philadelphia for the last six years. The principal objects of this day are:

(*a*) To arouse public interest in individual and community health, with a view to raising the vital index of the city.

(*b*) To emphasize the importance of health education, which should begin with the child of primary-school age.

(*c*) To call forcible attention to the importance of a strict observance of the rules and regulations of the Department of Health, which are intended to guard the public against the contraction of communicable diseases.

(*d*) To put the general public into possession of the better-known modern facts relating to individual and public hygiene, many of which are the result of experimental research.

(*e*) To crystallize public sentiment in the ways mentioned above in favor of the promotion of health by approved modern agencies and methods.

An educational campaign in the interest of the negro population was conducted in 1922 and 1923. In conjunction with the Philadelphia County Medical Society, Diphtheria Week was held March 12 to 17, 1923.

An active interest has been displayed in the elimination of odors, smoke and other nuisances related to the public health. Investigations have been conducted of industries handling hides, skins, hair and wool, with the view of preventing anthrax.

Better ventilation and sanitation in the street cars have been

obtained through the hearty coöperation of officials of the Philadelphia Rapid Transit Company. The moving picture theaters have also given their support toward the control of communicable diseases by displaying notices on the screens and improving sanitary conditions in the auditoriums.

A continuous health educational campaign has been conducted through the press, through the publication of the *Monthly Bulletin*, intended for the medical profession, medical social workers and the laity interested in health, and through the issuing of pamphlets on various health topics. Representatives of the Public Health Department have addressed lay audiences and carried their lessons of health directly to the public. A syllabus on hygiene and personal health has been introduced in the public schools. Individual instruction to mothers has been given by the nurses of the division of child hygiene. A plan for broadcasting health talks by radio is now under consideration.

In association with other municipal departments, there has been a keen, active interest in clean streets, clean-up week, protection of water supply from wells, control of the mosquito nuisance, enforcement of prohibition through chemical examination of confiscated liquors and various other miscellaneous activities.

The various offices of the department have been reorganized on a firm business basis, enhancing their efficiency and instilling a coöperative spirit in the service of the public.

The Department of Public Health has during the past three years devoted much time and energy in an endeavor to improve the hospital system of the city, which includes the Philadelphia General Hospital, Philadelphia Hospital for Contagious Diseases and the Philadelphia Hospital for Mental Diseases. While deeply appreciating the teaching facilities offered in these several institutions, it has been the policy of the department to instil a greater spirit of interest in the individual patient and greater consideration for the welfare of those entrusted to its care of paramount importance.

The medical directors and the staffs of the city hospitals have entered into the new order of things with enthusiasm and interest,

and have rendered to the city a service of incalculable value. The accomplishments of these institutions will now be considered *seriatim*.

1. Philadelphia General Hospital (1920–1923). Many changes have taken place during the past three years at the Philadelphia General Hospital, in its organization, administration and functions. Constructive changes and alterations have been quite numerous and were designed so as to give the maximum benefits to the sick and to the personnel of the institution. New departments have been opened and modern equipment has been supplied to meet every scientific advancement in medicine which may facilitate the diagnosis and treatment of disease.

Of great interest is the new Radiological Department, with its research laboratory in radiology, radium emanation plant and treatment rooms, which are open to the general public and afford treatment to those who heretofore were unable to secure this service because of the expense. This is the first municipal hospital in the United States to establish and own a radium emanation plant of such capacity and equipment. It is also reputed to be the only hospital in the city to have a modern high-voltage *x*-ray machine for the treatment of cancer and other diseases.

The neuropsychiatric department, formerly known as the psychopathic ward, has been completely reorganized. An outpatient department has been added where certain patients are examined before admission to the hospital and others transferred from various other departments suspected of having mental defects.

It maintains a traveling clinic, composed of a group of observers, who examine, study and treat cases of behavior abnormalities among school children. Expectant mothers are also examined in the neuropsychiatric clinic for mental disorders. The prevention and correction of early mental defects in these classes of patients is an intimate part of the general public health activity of the department. The hospital has also added a prenatal clinic for the observation of expectant mothers before admission to the hospital, a venereal disease clinic for men and women, a tuberculosis clinic for the benefit of residents in the immediate vicinity

of the hospital and a heart clinic provided with a modern electrocardiograph station for the study of cardiac conditions. The pathological laboratory is reputed to be one of the finest found in any hospital in the East. Its management and equipment are believed to be the best. In addition to aiding the correct diagnosis of cases, this laboratory is now being used by the graduate school of the University of Pennsylvania, where extension lectures are open to the general medical profession. Plans are under way for establishing private wards and rooms, for a metabolic ward, medical ward for city employees, a special ward for heart cases and an infirmary for residents and ex-residents.

Convalescent patients are no longer permitted to roam about idly in the wards or on the grounds of the institution, but are occupied by useful, light employment to arouse interest in work which will distract the patient's mind from his ailment. Workshops have been established where patients are engaged in weaving, carpentry, knitting, basket-making, molding, shoe-repairing and many other forms of light employment which will make crafty hands from idle fingers and create a happy state of mind from idle thoughts.

The management of the hospital itself is along modern efficiency lines, while the personnel of the institution is under instruction to give every possible care and efficient treatment to the patients and to exhibit a sympathetic feeling of interest in the welfare of the sick.

This personal touch with the patient has been augmented by the work of the medical social service department, whose duty it is to become intimately acquainted with the personal and family needs of every patient and to see that the speedy recovery of the patient is not jeopardized by home cares. After discharge from the hospital, patients are followed up by the medical social service workers, with the purpose of urging them to carry on after-treatment, and to assist the family personally and financially, through other agencies, to obtain a new start.

Time and the occasion forbid detailed mention of the many constructive changes and improvements in the physical plant on

the one hand and the demolition of certain old buildings in the center court, replaced by open gardens with trees and flowers on the other hand.

2. Philadelphia Hospital for Mental Diseases. On January 1, 1920, the Philadelphia Hospital for Mental Diseases had 2591 patients. On March 17, 1923, the census had increased to 2950. All of the insane patients housed in the out-wards of the Philadelphia General Hospital and at Holmesburg have been removed to the country branch of the hospital at Byberry.

In the spring of 1920 the opening of five new buildings of the female group at Byberry permitted the removal of these patients, thus relieving the congestion at the city branch.

The following, among many constructive changes, have been made at Byberry:

(*a*) Refurnished physical therapy wards for mental cases.

(*b*) Established workshop for occupational therapy in both male and female departments.

(*c*) Construction of infirmary for men and women.

(*d*) Increased output of farms.

(*e*) Increased personnel in the medical staff, nursing staff, nursing and physical therapy.

(*f*) Installation of hydrotherapy department.

(*g*) Established a dental service.

(*h*) Increased recreation and amusement facilities for all patients.

(*i*) Arranged separate dining room for quiet mental cases.

3. Philadelphia Hospital for Contagious Diseases. The principal recent advances at this institution may be conveniently tabulated thus:

(*a*) Construction, equipment and management of acute laryngeal diphtheria ward.

(*b*) Construction of intubation building for chronic diphtheria cases.

(*c*) Construction, maintenance and organization of convalescent ward and heart clinic.

(*d*) The adoption and construction of the cubicle system in all public wards, each containing twenty separate units or cubicles.

A system of modern plumbing has been installed in all the wards. This consists in knee-control washstands, bathing slabs and sprays for patients and utensils.

(*e*) Construction and operation of chapel and mortuary building, which are operated as separate units.

(*f*) Construction of a modern hospital for the care of smallpox cases was brought about by assembling and adding to the cottages formerly occupied by the minor contagions.

(*g*) The medical social service department was reorganized and facilities provided for interviewing patrons.

(*h*) The establishment of recreation and playgrounds for patients convalescing from diphtheria and scarlet fever (separate grounds for each).

(*i*) The construction of new wing in the laboratory of hygiene.

Activities of the Bureau of Health (1920–1922). The division of medical inspection has been very active in the prevention of smallpox by immediate hospitalization of smallpox patients and house contacts and by wholesale vaccination of persons in the affected districts.

The prevention of diphtheria has gained an added stimulus through the introduction and practical application of the Schick test and toxin-antitoxin immunization.

Several epidemics have occurred during the last three years and were controlled and abated quite effectively. In 1920 the epidemic of influenza, although severe in character, did not approach in intensity that which occurred in 1918.

Scarlet fever reached epidemic proportions in 1920 and 1921, while measles was widespread in 1922. A campaign of early detection and isolation was carried out, while children in the schools were instructed in the importance of clean hands in preventing the spread of these infections.

Disinfection by fumigation was discontinued in 1921 and there was substituted a thorough renovation and cleaning of infected households.

For four of the principal infectious diseases, comparison of the

death-rates for the last three years with a like period preceding is as follows:

	1917	1918	1919	1920	1921	1922
Typhoid fever . .	6.22	4.83	4.42	3.26	2.25	2.80
Smallpox . . .	0	0	0	0	0	0
Diphtheria . . .	25.64	21.91	24.45	22.74	17.30	14.83
Pulmonary tuberculosis	174.00	190.40	144.50	120.70	102.40	99.90

Division of School Medical Inspection. The principal activities of the division of school medical inspection include the annual physical examination of each school child, a yearly sanitary inspection of every school building, special examination of pupils suspected of contagious diseases and the medical supervision of children enrolled in the special classes of the Board of Education. An average of over 200,000 children were examined each year and physical defects were found at the rate of one for each child. The division was responsible for the correction of about one-half of the defects found. Greater care and attention to the school children have been made possible by the addition of forty nurses to this division, the personnel now being composed of eighty-three physicians and eighty-one nurses. Each physician and nurse have under their care 3000 children.

The sanitary inspection of the school buildings made each year includes ventilation, lighting, water supply and other sanitary features in each of the 300 school buildings, covering over 6000 class-rooms, 200,000 desks and 300 schoolyards and toilets. Considered, as a whole, the sanitary condition of the whole school plant is good and that of the newer buildings is excellent. The Health Department has been influential in raising the sanitary requirement in the schools.

The control of contagious diseases has always been an active function of this division, in that teachers are instructed to refer children who are ill to medical inspectors for examination. In 1922, 1635 cases of contagious diseases were detected in this way. Contact cases are examined, and in the case of diphtheria, cultures are taken to detect others in the incipience of the disease.

Children in special classes for the tuberculous, poorly nourished,

partly-sighted, crippled and deaf, to the number of 807, are also under the supervision of the school inspectors. In addition, about 1500 delicate children are gathered in groups of 25 for instruction in proper health habits, and about 400 quarts of milk are consumed by these children daily.

The division also has a clinic for the examination of mentally deficient children; for the examination of absentees for the Bureau of Compulsory Education; for physical examination of those children who apply for employment certificates and for candidates of teaching and other school positions.

Division of Child Hygiene. During the last three years the infant mortality was 82.8 compared with 107.5 during the three years previous. The lowest record was attained in 1921, when the mortality was 77.8. Two additional health centers were opened in 1920, making a total of ten in all. The activities of the centers have been extended to include heart clinics, nutrition classes, cooking and home-nursing classes for mothers.

Through the efforts of the division of medical inspection, Schick-testing clinics were opened in all the health centers, and these met with the hearty approval of the general public, as shown by the large numbers of mothers applying with their children for protection against diphtheria.

The nurses in the division of child hygiene visit about one-half of the babies born in Philadelphia and supervise their care until they are one year old. The mortality-rate of the babies under their direction is less than one-half of the total infant mortality for the entire city.

Children discharged from the Philadelphia Hospital for Contagious Diseases, particularly those recovering from diphtheria and scarlet fever, are followed up by the nurses of this division, with the view of having any physical defects corrected and keeping them under medical care and to prevent the possibility of physical defects which may arise following infectious diseases.

The division has supervision of boarding-houses for infants, private maternities, day nurseries, of the work of midwives and continues to distribute free milk to needy families.

Dental Dispensaries. There are now nine dental dispensaries in health centers and two in school buildings. They serve an average of 22,000 patients annually and perform, approximately, 92,000 dental operations each year. In conformity with the modern idea of dental prophylaxis as an important measure of personal hygiene the Bureau of Health has installed six portable chairs in one school building where dental hygienists are engaged to keep the teeth of children in a healthy state. When these oral hygienists have completed their work in one school they will continue to repeat the same service in other schools until all the children have been examined.

Eye Dispensary. Approximately 3700 cases are treated annually and spectacles are furnished free of charge to those who are unable to pay. Partially-sighted children are taken care of in special sight classes under the direction of the Board of Education.

Division of Food Inspection. The daily sampling and examination of milk have kept the milk supply of this city at a high standard of quality and comparatively free from the organisms of communicable disease.

Close watch is kept on all food-handlers, with the view of protecting food products against dirt and filth of the streets by requiring the covering of certain exposed foods and their elevation from the ground in conformity with the law. Meat and meat products are constantly under inspection. Approximately 4,455,000 pounds of unwholesome and diseased meats, fish and poultry were condemned, seized and sent to denaturing tanks during the last year.

Raids have been conducted along curb markets and large quantities of unwholesome food seized from dealers who seemed to congregate at points to take advantage of serving the poorer class of people with an inferior class of edibles.

A thorough inspection is maintained of all food products sold to city institutions.

Laboratory of Hygiene and Division of Chemical Analysis. Routine duties have been performed in examining specimens referred by physicians. Diphtheria antitoxin and typhoid vaccine are manufactured by this laboratory. Investigations have

been conducted for the department in the hide industries for the prevention of anthrax. During the summer of 1920 a systematic laboratory examination was conducted of rats in and about the city for the detection of plague.

In the division of chemical analysis all liquors and drugs were examined for the Police Department. A study has been made of fumes, odors and smoke nuisances.

Division of Housing and Sanitation. One of the principal accomplishments of this division has been the reduction of privy vaults on sewered streets from 8000 in 1919 to 345 as listed on March 12, 1923.

Piggeries have been entirely eliminated in restricted sections of the city.

The division has been actively engaged in the elimination of the fly, mosquito, rat and household vermin. Of the 9685 tenement-houses, rooming-houses and lodging-houses in 1922, only 23, or 0.2 of 1 per cent were found to be without any indoor water supply. This is a marked improvement over conditions existing prior to the enactment of the Housing and Sanitation law of 1915.

DISCUSSION

Dr. Alexander C. Abbott: Concerning the low birth rate, it is hard to say whether we have a low birth rate or not, but my conviction is that we do not know more than approximately what the birth rate is. There is a tremendous amount of carelessness among physicians in the matter of reporting births.

The rat campaign is, by some, thought to be amusing rather than serious. I can assure you that it is a vital question in every big seaport. The bubonic plague is spread by rats, and it is only by continuous anti-rat measures that we can keep the disease out of the country.

At a meeting during schoolmen's week recently one session was given over to the question of education in the elementary schools, and the belief was expressed that certain phases of personal and domestic hygiene, if presented by a master, could be made intelligible to children in the lower grades of school and should be part and parcel of their every-day education. I concur in that opinion.

During the last few years there has been a tremendous increase in the negro population in Philadelphia. I do not believe I am far from the mark when I say that the negro population has doubled in the last six years. This introduces a population factor that must be taken seriously. Among the negro population we find the question of birth reporting very carelessly handled; morbidity and mortality among colored infants is relatively neglected. It is among the colored population that we note a marked incidence of tuberculosis and syphilis. I think the time has come when we should invite the coöperation of the intelligent leaders of the negro community and lay the problem before them and help them to take care of it themselves. The problem is theirs as well as ours, and if they were properly instructed they could in part take care of it.

The question of anthrax may seem to us unimportant, but the fact that this disease may be, and usually is, contracted by the handling of hides makes it an item of importance in Philadelphia.

The street-cleaning work of the city is being reorganized on a logical basis. The officials realize that they are dealing with a large city and not a country town. The papers have had accounts recently of lawsuits over stables being erected for municipal use. I can assure you that all the city stables that I have visited are clean; there is no collection of manure about them, and every garbage cart is flushed with a fire hose each afternoon. This new idea of locating the stables where needed and districting the city is an excellent one. You can stand by the department in its movement for better service.

Concerning the provision for taking care of smallpox cases, I think this is preposterous, a totally unnecessary expense when everyone knows what will prevent smallpox. I believe that any city which has smallpox deserves every inconvenience connected with it, when we realize how easy it is to prevent it.

It is gratifying to know that during the last epidemic of scarlet fever the cases were all very mild and the mortality was almost negligible.

I do not wish to say anything discouraging, but we must realize that the great drop in tuberculous cases mentioned, from 174 in 1917 to 99.9 in 1922, is due largely to the fact that during the influenza pandemic many tuberculous people were carried off in one fell swoop.

Dr. A. A. Cairns: I agree with Dr. Abbott's remarks in regard to the erection and maintenance of a smallpox hospital, which would be a wasteful expenditure of the taxpayers' money. For the last twenty years under my supervision as chief medical inspector, 1,500,000 people have been vaccinated. I felt that Philadelphia was a well-vaccinated city, but during the last few years I have come to the conclusion that we are not as

well vaccinated or protected as we think we are, or as we should be. Last December 14 cases of smallpox developed in one street—7 in one house—with no medical attention (the original case came from Georgia); in the second house 7 other cases developed before the Bureau of Health was notified. Eleven other cases developed in other sections of the city, all having been in contact with the two houses mentioned. In offering vaccination to the inhabitants of the different areas quarantined on account of these cases, it was found that 20 to 40 per cent of these people were unvaccinated. I believe somebody is at fault in this matter of vaccination, and I do not know whether it is the doctor or the public; it used to be a routine matter for doctors not to discharge a maternity case until they had vaccinated the child—this, however, is not the case at present. There are a number of intelligent and well-educated people known as Christian Scientists who do not believe in vaccination. I find that medical schools have failed to teach the subject or technic of vaccination to medical students, and the majority of young doctors who graduate to-day have never done these vaccinations; if this is allowed to continue for the next twenty-five years we will have to teach vaccination all over again to students. Vaccination is not a difficult procedure, but requires a nice piece of technic.

Dr. Alexander C. Abbott (closing): I was lecturing recently before an advanced medical class in an institution whose name will remain unmentioned, and in the course of the lecture I asked if any of the audience had ever seen anybody vaccinated or if they had been taught how to vaccinate. I got no reply, and on singling them out individually and questioning them, I found that not one of them knew how to perform properly or at all a vaccination, so I taught them then and there.

SOME REMARKS ON MENTAL HYGIENE[1]

By EDWARD A. STRECKER, M.D.
PHILADELPHIA

Within the last twenty-four hours, 7000 children have been born in this country, and by a simple process of multiplication one is able to predict with confidence that the birth-rate for these United States during the year 1923 will be about two and one-half millions. Vital statistics are usually not of absorbing interest. It is popularly supposed that statesmen study them carefully, compare the birth-rate with the death-rate and according to the result of the subtraction either assure the common people that the nation is safe, at least for another year, or draw a sombre picture of national decadence and extinction. The entry of an infant into the world is usually heralded as a felicitous event. This is particularly true in this country, where the Constitution, with unintentional irony and somewhat grim though unconscious humor, insists that every child is born equal to every other child. Physicians, and perhaps chiefly psychiatrists, who by the nature of their calling may be somewhat inclined to a pessimistic viewpoint, cannot be quite as jubilant as the Constitution is about the future of yesterday's 7000 babies and this year's crop of two and a half millions. They know by reason of sad experience that many of the infant army will not even live long enough to appreciate the more or less deceptive equality guaranteed by the Constitution. They know that the early years, say from one to twenty, are like so many corners around which lurk enemies who strike to kill. Infantile and childhood diseases, tuberculosis and syphilis, malaria

[1] Read May 2, 1923. The quotations in this article are from Dr. Frankwood E. Williams, Community Responsibility in Mental Hygiene, Amer. Review, January-February, 1923.

and hookworm, and a host of other morbid conditions all claim their toll. At this point it is presumed that you are all properly steeped in gloom, and as a student of psychiatry I may contribute the cheerful information that 1 of every 26 of the babies will eventually become insane. To make the figures somewhat more impressive, it may be added that each year 50,000 individuals are admitted to state hospitals for mental diseases. Many more should be, but that is another story. I do not know the number who are deficient or become otherwise mentally and nervously handicapped, psychopathic, psychoneurotic and the like, but I know the percentage is high.

The time has arrived to sound a more optimistic note. I know of no surer way of accomplishing this than by asking you to reflect on the splendid strides which have been taken by the public health movement, the seven league boots in this instance being furnished by scientific medicine. The number of foes who lie in wait for the 7000 children who were born yesterday has been appreciably diminished. We may only recite a few outstanding examples: Vaccination struck a death blow at smallpox, and today only the fool or the unfortunate child who has fools for parents need have this dread disease; there is a specific for diphtheria and an almost specific for syphilis; there is effective life-saving treatment for malaria and hookworm; sanitation has made typhoid a rare fever and tuberculosis is under better control. When a child develops a contagious disease, other children and the community are protected by an elaborate municipal system of supervision and isolation. During the school age, children are routinely examined physically. They are taught how to walk and how to sit; how and what to eat and what not to eat; when and how to sleep. Their physical handicaps are corrected; their bodies are straightened and strengthened; their vision, if defective, is aided by lenses, and their teeth are kept in good repair. I might continue to chant this pean of gladness for hours. However, I would finally come to the end of the song, and then, as a psychiatrist, I would want to finish with this glorious climax—and the minds of the children are carefully and scientifically studied and the laws of

mental hygiene are applied—but my climax would be a ludicrous anticlimax, since it would not be true.

Don Marquis, in *The Revolt of the Oyster*, tells the tale of the great fight for supremacy between Probably Arboreal, the representative of our early ancestors, and the giant oyster. "As Probably struggles with the unknown thing that holds his foot as in a vise, he calls for help. But while his friends gather about on the shore to watch the combat, which is to end apparently in the drowning of Probably, none will help, for they are sure it is a god or a devil or an octopus that has Probably Arboreal in its clutches. Since spectacles are always interesting, they sat down comfortably on the beach to see how long it would be before Probably Arboreal disappeared. Gods and devils, sharks and octopi, were forever grabbing one of their number and making off to deep water with him to devour him at their leisure. If the thing that dragged the man were seen, if it showed itself to be a shark or an octopus, a shark or an octopus it was. If it were unseen, it got the credit of being a god or a devil." After all, we have not traveled so very far since the time of Probably Arboreal. We advance boldly to give battle to the sharks and octopi which may be seen, but are apt to give the unseen gods and devils a wide berth. In this connection the sharks and octopi may be said to represent the causes of physical disease like tuberculosis, syphilis, malaria or typhoid, which may be captured and even viewed through the microscope, while the gods and devils are comparable to the things which produce mental disease or deficiency or neuroses or allied states. The majority of these things have evaded discovery, and perhaps this is the reason why physical hygiene has outpaced mental hygiene. Of course, the former also has had the advantage of a much earlier start.

It is probably true that sometimes psychiatrists themselves have beclouded the issue and made out a rather poor case for mental hygiene. When there are so many practical problems in mental hygiene, it seems rather inconsequential to speak of introducing psychoanalysis into the public schools, or, as in a book which I read some time ago, to recommend that noise of machinery

be studied so that it may be attuned to the innate rhythm of the factory workers.

I intend no reflection on psychoanalysis or on those who are seeking to make the life of the laborer more esthetic, but merely cite these two instances as types of things which should at least be deferred until the obvious is laid hold of and the more glaring examples of bad mental hygiene have been corrected. Therefore, the remainder of these remarks will be restricted to little more than a bare enumeration of certain considerations, hard and sometimes ugly facts, which demand attention almost as urgently as does an epidemic of smallpox.

There is nothing vague or visionary about the problem of the feeble-minded child in the public schools. He is there in every school and in every class-room, and yet economically and morally it is wrong for him to be there. His mind has not been designed to profit by a system of education devised to meet the needs of the average child, and any development along mechanical lines which might be possible for him is lost forever. Furthermore, his presence in the school-room, where he consumes a tremendous amount of the teacher's time, all to no useful purpose, is as unfair to the other children as it would be to ask an athlete to run a race with weights fastened to his legs. It is scarcely worth while for the psychiatrist to point out the defective child, for it is only with great difficulty that an occasional one may be admitted to the proper institution, and usually only after a long delay. The majority of these unfortunate children will be permitted to grow to maturity and originate long lines of feeble-minded progeny. If only mental deficiency had a skin rash as one of its symptoms, then the law might step in and placard the house in which the patient lives, and thus warn the community of its great danger.

Mental hygiene asks a fair hearing for mental disease. There is nothing unusual or extreme about such a request. I mentioned that at least 500,000 human beings will become insane in the United States during the year 1923. I do not know whether the number of individuals who will fall ill with typhoid, malaria, tuberculosis, cancer, heart disease, appendicitis and a host of other evils to

which the flesh is heir will be twice or ten times or a hundred times as great, but I do know that they will practically all of them receive reasonably careful and scientific study and treatment. No matter whether they be millionaires or paupers, or whether they live in a great city or a small town or even a hamlet, they will be able to find fairly close at hand splendidly equipped and staffed hospitals where every resource of modern medicine will at once be enlisted in combating the disease from which they are suffering. Should they be unfortunate enough to have some baffling and incurable ailment, like inoperable cancer, they will at least be spared unnecessary pain and perhaps be able to find vicarious satisfaction in the thought that even if their lives cannot be spared, yet science, supported by enormous sums of money, both public and private, is studying and laboring to find the solution of the problem, so that future generations may be saved.

How different is the situation for the man who becomes insane. Only if he is particularly fortunate will he be placed in a hospital where there are even average facilities for care and treatment. An overwhelming proportion will only be given rather unsatisfactory custodial attention, and for a not inconsiderable percentage there will not be any treatment at all. There is a psychosis called dementia precox which may be compared to cancer, in that once it gains a firm foothold usually only death can give release. In neither instance is the cause understood, but here the similarity ends. Consider the wonderful institutions, temples of research, where every day bands of devoted workers, supported by humane philanthropy, are delving deeper and deeper and getting closer and closer to the ultimate origin and nature of carcinoma, and then picture the back wards of public mental hospitals filled to overflowing with hopelessly demented creatures with only here and there, very few and far between, a few humble laboratories with students striving against odds to solve the riddle and forced to proceed very slowly for lack of funds. It may be granted that dementia precox is a baffling ailment, but so is cancer, and surely we will all concede that they are both diseases and should be investigated. Surely we will not continue to be dominated by the thoughts

which were in the sluggish minds of the friends of Probably Arboreal, as they sat on the bank and watched his struggle to free himself from the clutches of a mighty and unseen force. If this archaic idea still holds, then we will feel that since in cancer there is something tangible—something to be seen and touched and handled—therefore it must be due to a shark or an octopus which may be caught and destroyed; but in dementia precox, since the thing which is dragging a mind below the surface is invisible, it must be either a god or a devil, and there is nothing to be done about it.

As a matter of fact, the causes or at least the chain of events which may lead to the development of mental disease is not quite as obscure as is commonly believed. If the links which make up this chain are separately studied it will be discovered that at least some of them may be identified and probably gradually eliminated. This is real hard-pan, practical mental hygiene. It is popularly supposed that an individual goes insane suddenly—somewhat akin to the explosion of a bomb—but psychiatrists believe, on the contrary, that the process is slow and that a psychosis is a long time in the making. Sometimes, indeed, its beginnings reach far back and may antedate the patient by one or more generations and be traceable to his father or his grandfather. Personally, I believe the effect of heredity has been somewhat overrated, but, nevertheless, it is far from being a negligible factor. Drastic laws aimed at this factor will not accomplish anything, but molding of lay opinion by legitimate and sensible propaganda will eventually bear fruit.

Personality is scarcely definable, yet we know, in a general way, that it is the end-result of everything that has gone before, including inheritance, and that it is further largely shaped by environment. From a study of the surroundings in which many children live one is almost tempted to remark that often, instead of shaping, the environment hammers the personality out of shape. The primary elements of the psychology of child-life—suggestibility, imitativeness, love of power, curiosity, some degree of reasoning ability, etc.—are potent forces for good as well as for evil. Average, sensible adults, by dint of hard experience, must necessarily come

to some conclusion concerning those traits which are needed in the struggle for existence and those which prove to be handicaps, and yet how comparatively rare it is to find any concerted and consistent effort looking toward character formation in children. It is not at all unlikely that a certain proportion of mental disease arises from the social incompatibility which results when a crippled personality is brought into contact with stern reality.

May we ever put our fingers on a definite, concrete agent—in other words, a real shark or octopus—and say if this had not been present mental disease would not have developed? Mental hygiene points out such instances and suggests that they be controlled. One thinks at once of paresis and other forms of luetic insanity for whose development infection by the spirochete is a necessary condition. There is a double chance of avoiding these psychoses which make up about 10 per cent of all mental disease. The first opportunity lies in the reduction of the amount of syphilis itself; the second, in the active and thorough treatment of its initial stages. Certainly we have not made the maximum amount of effort in either direction. Syphilis is as much or more of a menace to the community as is any contagious disease, and needs to be brought under the same rigid public discipline.

A considerable percentage of mental disease is due to poisons taken into the body, over a long period of time, either deliberately or accidentally. In the former group belong alcohol, and perhaps particularly the kind of alcohol which has been circulated in certain quarters since the passage of the Volstead act, opium and its derivatives, cocaine and many other toxic drugs. In the latter group are included the industrial poisons, such as lead, mercury and silver. In both instances more effective protection is possible and should be enforced. It is true that there is a certain nucleus of confirmed habitues who are beyond reach; but more important from the standpoint of mental hygiene is the much larger fringe who begin as onlookers or amateur experimentalists and in whom the experiment is completed before they realize it. For them a psychosis may rest on the ease or difficulty of access to drugs. In the industries, simply as a common-sense proposition of man power

conservation, every precaution should be taken to guard the workers against chronic poisoning.

For many years there have been two armed camps in psychiatry —the organicists and the functionalists. The first camp believed that mental disease always had a strictly physical basis, and the second maintained that it was occasioned solely by psychogenic instrumentation and was without organic changes. For a long time each frontier was jealously guarded and there were many bitter quarrels; but, finally, certain outsiders, chiefly the experimental physiologists, stepped in and patched up a fairly satisfactory truce by proving that both sides were more or less right. Examples, to the effect that the mind may dominate the body, and *vice versa*, may be freely quoted. They are so old that they have almost been forgotten. For instance, the conditions spoken of, cretinism and myxedema, are easy to recognize, and it is known that they are caused by a disturbance of the thyroid gland. In addition to definite physical symptoms there are also mental phenomena, notably a sluggishness of the mental processes and an upset of the emotional balance. Here is a simple illustration of how somatic disease may involve the mind and produce abnormality. Likewise fever, when it mounts to a high point, is in itself sufficient to unbalance even the most stable nervous system. Delirium is a psychosis. The reverse, or the power of the feelings and emotions to command the body, is just as readily demonstrated. If one eats a meal while being dominated by a strong, moving emotion, such as anger or fear, the digestive processes will be interfered with and there will be gastrointestinal symptoms. There are well-authenticated reports that fear may even produce death. In some of the South Sea Islands where voodooism is still practised, natives die in a few days after they have been told that a small wax image of themselves has been properly cursed and melted in the fire. Picked athletes—in fact, members of a 'varsity football squad—have repeatedly had sugar in the urine on the eve of their most important match. You will recall some of the more common laboratory experiments. The dog when shown food begins to secrete the digestive juices. By combining the

sight of the food with the ringing of a bell for some time, we gradually come to the point where the sound of the bell alone suffices to inaugurate the digestive function. Again, if a cat is frightened by a fierce dog, there will be an outpouring of adrenalin which may be demonstrated by physiological chemical methods.

It is a long step from the experimental animal to the highly complex human being, but I have two patients in mind who bear out in a crude way the possibility of a somatopsychic or psychosomatic mechanism. They are both women, and they both have manic-depressive psychoses.

The first patient was subjected to an unusual combination of somatic strains at the age of thirty-one years. Coincident with a severe attack of scarlet fever there was an abortion, and as immediate sequels, septic infection and marked kidney damage. For years this patient has had regularly alternating periods of excitement and depression. The change from one phase to the other is so complete that it is hard to realize that she is the same person. This in itself is not remarkable to psychiatrists, but the periodic blood-pressure alternation which accompanies the contrasting emotional states is noteworthy. During the melancholic period the systolic pressure is about 170 or 180, but with the onset of mania it jumps to 210 or even 250 or more, to remain at a high level until the approach of the next depression, when it declines 50 or 60 or more degrees.

The psychosis in the second patient appeared at the age of forty-eight years, following immediately the death of her husband. They were devoted to each other, and after he became infected with tuberculosis she nursed him constantly for ten years, always refusing to believe that he could not be cured. It was noticed that during this prolonged period of stress and worry the thyroid gland enlarged from time to time and there was other evidence of toxic thyroid manifestations. The same symptoms of endocrine dysfunction persisted during the psychosis.

These two cases are briefly quoted because they seem to establish the working of a vicious somatopsychic circle. No one would dare decide whether the psychic or somatic element came first, but it

does seem reasonably sure that at some time before the insanity came to the surface there was a reaction between emotional and physical components, and they continue to set each other into motion in a pathological manner.

It may appear that I have traveled far afield from the subject of mental hygiene, but in reality there is hardly anything more pertinent than the effect of organic strain and psychogenic stress on the mental health. In the struggle for existence, which is now so complex and intense, there are, I think, periods in every individual's life when the margin of safety becomes diminished to a dangerous point. The factors which may mean the difference between sanity and mental disease are still obscure, but they are no longer mysterious or supernatural and their outlines are beginning to emerge from the shadows. This means that eventually they will be brought into the light, and who can say that finally they may not even be identified in the test-tube or under the microscope?

I have indicated very inadequately some of the more obvious problems in which mental hygiene is interested. They were mental deficiency (particularly feeble-mindedness in children) and heredity, the handicaps under which scientific psychiatry labors, being denied the ways and means which are freely accorded to other departments and specialties of medicine, the dynamic force of the personality, with the possibility that it may function as a destructive or constructive power and the identification of a few of the agents or conditions which may pave the way for the occurrence of mental disease.

If there is any moral to these few remarks, it is that psychiatry is simply a branch of medicine, and insanity is merely disease like any other disease. In other words, there are no gods or devils, only sharks and octopi. Once this is thoroughly realized by an intelligent public there will be a whole-hearted, almost universal move in favor of mental hygiene. To complete the tale of Probably Arboreal: "Already there is a stir among Probably Arboreal's friends on the shore, for Probably has reached down and grasped the thing which has him by the foot, and with a mighty

effort has thrown himself backward towards the shore. As his feet came out of the water they see it is an oyster, a giant oyster, but an oyster that has him by the foot—neither a god nor a devil, not even a shark or an octopus, but an oyster! In the allegory Probably wins, and the race of oysters sinks into the sea, never again to challenge." Don Marquis does not say, but it is likely that there was much hunting down of oysters after this incident.

DISCUSSION

Dr. Albert C. Buckley: Dr. Strecker has very clearly and ably indicated the mental medicine program necessary for the education of the entire public, including the layman, family physician, the jurist, etc., in fact, everyone. I think we are entirely safe in taking the stand that if mental medicine had progressed without any obstacles from the time of Hippocrates to the present day, we would be spending just as much money and effort and time in the field of investigation in mental medicine as in any other department. Unfortunately, there came the period of the Dark Age when the pendulum swung back to superstition and everyone believed that mental disease was caused by evil spirits coming into the body of the patient, and ever since that we have been trying to blot out that idea. There is a remnant of superstition in everyone of us at the present time; the truth is particularly brought home to us only when it comes to some member of our immediate family or our own friends or patients. Then we exert our efforts for the salvation of this patient from mental enfeeblement. We lack time as general practitioners to give sufficient thought to the situation and therefore do not take to heart the need for activity from the standpoint of early study and treatment of the mental patient. The prevailing thought is that matters dealing with mental disease are beyond the pale of tangible, substantial things. We are apt to see only the mental disorder of the patient when in reality he is a sick man. Dr. Strecker has indicated that there is a hopeful outlook; it grows more hopeful every year and the general practitioner seems to be growing interested in the mental patient. It is not to be expected that there is going to be a general turnover or a great revolutionary change in the methods of study and cure of mental disorders, but we are going to continue to study patients as individuals and as medical problems and find out, where possible, what is the matter with them and remove the exciting factors if possible. An internist well known in this city, whom I happened to meet recently in

consultation, said, "I believe you are curing as many patients in your hospital as we are in the general hospital:" I said, "Yes: taking all things into account, the percentage of actual recoveries of the patients in the general hospitals is not very much different from the recovery rate in mental hospitals if you exclude the accident patients and include carcinoma and all chronic diseases. I think we have reason to be hopeful, and we certainly need the aid of every medical man, irrespective of what his interest and specialty may be."

THE USE OF INSULIN IN THE TREATMENT OF DIABETES MELLITUS

The Nathan Lewis Hatfield Lecture

Lecture V[1]

By FREDERICK GRANT BANTING
TORONTO, CANADA

The lecture this evening will deal more particularly with the clinical application of insulin in the treatment of diabetes mellitus. Reviewing the outstanding contributions to our knowledge of the disease, one finds in the very early writings descriptions of its classical symptoms. It was known even at the time of Moses, but Paracelsus was the first to carry out clinical experiments. At the time of Paracelsus the disease was known, had been described and had its name. It was simply a disease characterized by the three cardinal symptoms of increased appetite, increased thirst and increased urination, with progressive loss of weight, of seemingly bad prognosis. Paracelsus evaporated a quart of the urine from a diabetic patient and found that it contained four times the amount of sediment that is contained in the normal urine. He described this sediment as salt, but it remained for Dobson, later on, to show that this salt substance was analogous to sugar.

It was not until the time of von Mering and Minkowski, in 1889, that diabetes was definitely connected with lesions of the pancreas, and since that time a very great deal of physiological investigation has been carried out in the endeavor to work on the suggestion of Lepine, that the experimental disease as found

[1] Read June 6, 1923.

in depancreatized dogs was due to a missing substance, an internal secretion, which both clinical and laboratory workers endeavored to isolate and to administer for the alleviation of the symptoms of the disease. Zuelzer tried pancreatic preparations extracted with alcohol on five patients and met with some success. However, Zuelzer's extract contained so much toxic material that the deleterious effects overbalanced the clinical improvement and Zuelzer abandoned the treatment. Pancreatic extracts were then lost sight of, principally because Forschbach, working in the clinic of Minkowski, was not able to substantiate the beneficial effects of Zuelzer.

From that time, 1908–1910, the treatment of diabetes mellitus took a distinctly different course. It was found, beginning more particularly with the contributions of Guelpa, Allen, Joslin, Woodyatt and a number of others, that the best results were obtained by a strict dietetic régime. Had a remedy for diabetes been found of value at the time of Zuelzer, medical science might have lost a great deal of its knowledge of the value of food in metabolic diseases.

The principle underlying dietetic treatment was directed at decreasing the total caloric intake, but principally reducing the amount of carbohydrate ingested. There is one other characteristic of dietetic treatment, namely, the low percentage of carbohydrates in the foodstuffs ingested. The rationale of the low percentage of carbohydrates is found in the fact that such substances as 5 per cent vegetables digest more slowly and absorb more slowly, so that the normal mechanism is able to look after a larger quantity of glucose.

The pancreas consists of two distinct types of cells, the acinous and the islet cells. It seemed reasonable to think that the failures on the part of former observers might be due to the fact that they did not attempt to get rid of the products of the acinous cells. Villiard and Arnazon tied the pancreatic duct in dogs, in 1884, and described the degenerative changes which took place in all the acinous tissues. The acinous cells are replaced by fibrous tissue, but the islet cells remain intact and the animals do not

develop diabetes. It seemed then we could get rid of these acinous cells and proteolytic substances.

Work was begun in the University of Toronto with Mr. C. H. Best. We first tied the pancreatic duct in dogs and waited from seven to ten weeks for degeneration to occur. We examined that material histologically and found, for the most part at least, it contained fibrous tissue and healthy islets. This material was chopped into fine pieces, put into ice-cold Ringer's solution and frozen. It was then ground up, extracted and finally filtered until clear. The antidiabetic properties of this extract were tested on depancreatized dogs.

Depancreatized dogs, as you know, within twenty-four hours develop a very high percentage of sugar in the blood and pass large quantities of sugar in the urine. There is a rapid absorption of subcutaneous fat, causing extreme emaciation in the course of five or six days. As a rule, their wounds do not heal and frequently a scum gathers in the corners of their eyes, and the dog is the picture of dejection. It was found that on the administration of the material made from the degenerated pancreas of duct-tied dog there was a reduction in the percentage of sugar in the blood, within two or three hours the blood sugar could be rendered normal, the urine became sugar-free, and, most characteristic of all, there would be a marked improvement in the general clinical condition of the dog. We have had dogs so weak that they were unable to stand up when raised to their feet, but two or three hours afterward would be standing up in the cage, wagging their tails and behaving as normal. They licked their wounds, and the wounds healed kindly when the blood sugar was normal. This was the first type of extract used.

The next type was very temporary: It was obtained from the exhaustion of the acinous cells by the long-continued injection of secretin; but it was almost impossible to totally exhaust the acinous cells and these extracts contained varying amounts of toxic substances.

The next type of extract was that obtained from fetal calves. It was known that other glands of internal secretion contained

their characteristic content as soon as they were differentiated in their embryological development. It seems reasonable to think that since the body does not require the external secretion of the pancreas until digestion is necessary, namely, after the time of birth, that it would not be present in the embryo. We later found that Ibrahim was unable to find the presence of proteolytic enzymes in the pancreas of the fetus under four months' development. Extracts prepared in cold Ringer's solution of fetal-calf pancreas were found to have the same properties as degenerated gland extract. Their injection was followed by a rapid and marked reduction in the sugar of the blood. The dogs became sugar-free, there was marked clinical improvement and no toxic symptoms. That the internal secretion of the calf, or extract prepared from the fetal pancreas of calf, exerted an effect upon depancreatized dogs was the first indication that we had that this substance was universal in the animal kingdom. We were able to obtain a number of fetal calves per day, which supplied us with extracts for more rapid physiological investigation. We were able to extend the study and to show that the active principle could be dissolved out with alcohol, and that it was precipitated with 95 per cent alcohol, and that it was soluble in everything up to 65 per cent alcohol, and, with the continued administration of this type of extract, depancreatized dogs could be maintained alive for a much longer time than usual. We were able to prolong the life of a depancreatized dog for seventy days, at which time it was able to run the floor. The dog was chloroformed and autopsied to prove that it was depancreatized.

Our great problem at this time was to obtain chemical means for the extraction of the active principle of the whole pancreas of the animal, because this is the potential supply. Extracts were made of 60 per cent alcohol, using the whole pancreas ground through a meat-chopper. The filtrate from this mixture was filtered until clear and then evaporated by electric fan and warm-air current and the residue distilled in saline or distilled water. This extract produced marked blood-sugar lowering effects.

At this time Dr. Collip was associated, and he ran the scale of

fraction of precipitation between 65 to 95 per cent of alcohol and succeeded in precipitating the active principle. Following along the history of insulin, various improvements and more and more delicate refinements have been made in the process of manufacture, until now insulin is fairly well standardized as to potency. Nearly all the toxic materials have been eliminated.

Possibly it would be well to review briefly some of the outstanding physiological experiments carried out in Prof. MacLeod's laboratory. It was found that glycogen could be stored in the liver of the depancreatized dog. The depancreatized animal has lost the ability to store his glycogen. It was found that the respiratory quotient of these depancreatized dogs could be markedly raised, indicating that the animal could burn carbohydrates. Other forms of hyperglycemia, such as suffocation and carbon-dioxide poisoning, could all be controlled by adequate doses of insulin. Fat, which is found very abundantly in the liver of depancreatized dogs, could be diminished in amount. Therefore, we concluded that insulin had some part to play at least in the mobilization of fat.

The clinical investigation commenced very early, but of necessity was succeeded by physiological investigation. The first clinical tests were carried out by Drs. Campbell and Fletcher, under the direction of Prof. Duncan Graham. It was found that severe diabetics could be rendered sugar-free, that acetone bodies could be markedly reduced or eliminated and that there was marked improvement in the clinical condition of the patient. Unfortunately, however, in transferring from small- to large-scale production difficulties were encountered and clinical investigation had to be abandoned. It was reëstablished at the Christie Street Hospital for Returned Soldiers, in association with Dr. Gilchrist. A little later the work was taken up again in the Toronto General Hospital. The results have been collected from all these sources.

The general management of the patient admitted to the clinic is as follows: The patient is first placed on the same diet as previous to admission, unless contraindicated by severe acidosis or

coma. They remain on this diet for about twenty-four hours, in order to gain some idea of the severity of their case and prevent too rapid change in diet. The patient is then placed on a basal diet, estimated from the Du Bois chart and Aub Du Bois table. This diet is planned to contain enough protein to maintain nitrogenous equilibrium, and carbohydrates and fats in such proportion that ketone formation will be prevented. The ratio of foodstuffs is approximately that of Woodyatt's formula. Patients remain for six to ten days on this basal requirement diet, during which time a careful history is taken, with particular attention paid to the history of heredity and infection. Careful physical examination is made, attention being paid to possible foci of infection. It is usually found that at the end of three or four days on this well-balanced basal requirement diet the daily output of glucose in urine becomes fairly constant. This is of value because the dose of insulin which we will finally give is estimated from the average daily output for the most part. During this time, the object is to gain as accurately as possible an idea of the severity of the case. If the patient becomes sugar-free on this basal requirement diet, the diet is raised. It has been our policy not to administer insulin to patients who are able to metabolize 700 or 800 calories over and above the basal requirement diet, that 700 or 800 calories being sufficient for his ordinary work. However, if the patient does not become sugar-free and is unable to take 600 calories above his basal requirement, he is placed upon insulin treatment.

Insulin is administered subcutaneously once, twice or three times per day, according to the severity of the case. As a rule, we give insulin twice a day, before breakfast and before the evening meal. It is given about twenty minutes to a half-hour before mealtime, in order that the insulin will be absorbed from the site of injection at the time that carbohydrates are being absorbed from the gastrointestinal tract. The amount of insulin administered is estimated in units. A unit of insulin was arbitrarily taken as the amount of active principle required to reduce the blood sugar of a normal 1 kg. eighteen-hour starved rabbit from its

normal level of 0.123 per cent to 0.040 per cent. This amount of active principle, in severe diabetics, accounts for about 2½ grams of glucose.

In cases complicated by an infection and which have a temperature there is not so much glucose metabolized per unit, possibly 1 gram per unit. In less severe cases there is more—possibly from 5 to 6 grams—metabolized per unit of insulin. There is, furthermore, considerable variation in the individual response to insulin. In some patients there is more marked lowering of blood sugar and a greater amount of metabolism per unit than in others. It is therefore safer, in commencing insulin treatment, not to endeavor to render the patient sugar-free immediately, but to give small initial doses and increase to the required amount. There were two patients in the same ward of about equal severity. One had a blood sugar of about 0.3 per cent and the other 0.32 per cent. They were approximately the same weight and size. Each was given 20 units of insulin. The patient with the higher blood sugar became unconscious from overdosage, while the other patient scarcely became sugar-free. It is therefore safer to commence treatment with 5-unit doses and rapidly increase until the blood sugar becomes normal and the urine free from sugar. The general management of patients, once rendered sugar-free, is simply the balance between artificial insulin and diet unaccounted for by the patient's own pancreas. In other words, sufficient insulin should be given to account for the diet over and above his own tolerance.

If too much insulin is given there is a symptom-complex characterized by increase in pulse, which is especially prominent in children. Pallor is marked in some children, flushing in others. Perspiration is frequently a marked sign. The most characteristic features of the hypoglycemic reaction are the patient's feeling of tremulousness. Even a little child of five or six years of age will tell its mother that they feel trembly. Adults express their sensations as tremulousness and fear of impending danger, and this, once experienced, is so striking that it is not overlooked on a second occasion. If patients are untreated the blood sugar may

go lower. The patient becomes incoherent in speech, finally goes on to drowsiness and unconsciousness, and this unconsciousness is sometimes intermittent with convulsive seizures. The same condition was formerly observed in rabbits who had received an overdose. The level of blood sugar at which these hypoglycemic symptoms occur varies a great deal. It depends not only upon the extent of fall, but also the rapidity of fall. When a patient has a high blood sugar, let us say 400 mg., and the blood sugar is rapidly reduced, he will experience symptoms when the blood sugar is at the level of possibly 0.15 per cent, whereas the same patient when he has been rendered sugar-free by treatment will not experience hypoglycemic sensations until his blood sugar is 0.07 or 0.06. Hypoglycemic reactions are dangerous, especially at night, because we fear that a patient may have a severe reaction and become comatose during sleep. We have not yet, however, observed untoward results, since our nurses are instructed when patients are commencing insulin to make frequent observations at night.

Fortunately, treatment of hypoglycemic reactions is readily administered and can be given by the patients themselves. The cause of the reaction is the lowered percentage of sugar in the blood. The treatment, therefore, consists in raising the percentage of sugar in the blood. This can be done by administering glucose in practically any form—cane sugar, orange-juice, glucose or food. However, the best form and most convenient for the patient who is about his work are these glucose candies wrapped in yellow paper; they weigh from 2 to 3 grams and may be constantly carried about by the patient. If the patient is unconscious with hypoglycemic reaction the blood sugar can be most advantageously raised by 10 to 15 minims of epinephrin. This causes sufficient glycogen to be released from the liver to raise the sugar in the blood and relieve symptoms. Epinephrin should be followed by glucose when consciousness is regained, to maintain the blood sugar.

The object of insulin treatment is to balance between the hyperglycemia produced by food, on the one hand, and the

hypoglycemia produced by insulin, on the other. The time of administration is an important element. It is given from twenty minutes to a half-hour before meals, in an endeavor to superimpose the curve of hyperglycemia produced by the meal upon the curve of hypoglycemia produced by the insulin. If this balance is carefully maintained, I believe it possible to keep the most severe diabetic sugar-free on a diet in which he can carry out his work or a diet amounting to twice his basal requirements. When this balance of insulin and diet is obtained we gradually increase both, keeping them parallel until the patient is taking around 2400 to 2700 or even 3000 calories. Mild cases receive one injection of insulin per day, before breakfast, More severe cases receive two doses, one before breakfast and the other before the evening meal.

The type of patient we have had has been the thin, emaciated, underweight type, and it was advantageous to give these patients a diet not only to increase their strength but to allow some gain in weight. When patients receive this increased diet they gain very rapidly in strength, weight and vigor. The most marked thing observable is the change in mental attitude of the patient. From a pessimistic disposition, with no outlook for the future, the patient becomes bright, happy and optimistic. It is particularly pleasant in the case of children, because they are very often underfed and without sufficient strength to play and run about as normal children. We therefore endeavor to maintain our patients sugar-free on a diet amounting to possibly twice their basal requirement.

In children it is often difficult to maintain the balance between increased food and insulin, because the margin between hyperglycemia and hypoglycemia is narrow. In adults where there has been a high blood sugar for a long period of time the kidney threshold is often much higher than normal, consequently it is much easier to maintain them sugar-free.

We cannot say at present whether insulin will permanently raise the tolerance to carbohydrates of the diabetic or not. Certainly, these cases that have high blood sugar for long periods of

time, and have been constantly excreting large amounts of sugar in the urine, show marked improvement in tolerance after being sugar-free for a few weeks. It will take considerable length of time to find out the ultimate results in the cases under treatment, but there is no doubt that there is a marked improvement in tolerance of a great number of patients—not, however, in all. Many cases require a progressively decreasing dose. Some patients who formerly required as high as 60 to 80 units to maintain an adequate diet are now able to remain sugar-free on half that amount.

A very severe diabetic came to Toronto on August 16 last. Sugar had been first noticed in the urine in April, 1919, and at that time she weighed 85 pounds. She was placed upon a diet and was very carefully treated, and was maintained sugar-free. The diet had to be gradually decreased from time to time, and on arriving at Toronto she was extremely emaciated, weighing only 45 pounds. She had also decreased half an inch in height, not having sufficient tone in her back muscles to stand erect. Her hair was dry and thin; her skin was wrinkled, scaly and very atrophic, and the subcutaneous tissue was almost completely absorbed; she was so weak that she could scarcely walk about the room without the aid of a support. Her diet was increased from 900 calories per day to 2700 calories, and was so balanced with insulin that she remained sugar-free for three months, except for eight days, when sugar was present in traces in the twenty-four-hour specimen. At the end of this time she had gained about 28 pounds in weight. She was able to take strenuous exercise, walked and ran about and was greatly improved in every way. Insulin was then discontinued and her old diet resumed. It was found that she had neither lost nor gained tolerance.

However, in contradistinction to this case there is one, a child of fourteen years of age, in whom sugar was first noticed in the urine four months previous to admission. When sugar was noticed in the urine she was placed on a diet which, although abundant, the child did not adhere to, but ate bread and candy when she got the chance. Sugar appeared time after time. Finally

she ate a large number of grapes and olives, and within a few hours had pain in the stomach, vomited, became very sleepy and finally became comatose. She was admitted to the hospital twelve hours after the onset of these symptoms. Her blood sugar was 0.5 per cent; she had a 4-plus acetone test of urine; there was a distinct odor of acetone on the breath and she was sufficiently anesthetized in coma to permit cutting down on a vein in her leg without anesthetic, showing she was completely comatose. The child was given 40 units of insulin at 3 o'clock in the afternoon, about an hour after admission. At 5 o'clock, or two hours after the injection, the blood sugar had fallen to 0.2 per cent. At 7 o'clock it had further declined 0.1 per cent, and at that time we became alarmed that she would show a hypoglycemic reaction. She was recovering somewhat from coma, but still unconscious. She was given 17½ grams of insulin, and at 9.00 P.M. she was sugar-free, acetone-free and the blood sugar was 0.08 per cent. Her clinical condition changed at this time. She became conscious, but as she became conscious went into a muttering delirium. Her temperature became rapidly elevated to 101°, then 102.5° and 103°, and at 12 o'clock that night temperature had reached 105°. We had given an enema, but with little result. The chest was repeatedly examined for pneumonia, but no signs could be found. With the increase of temperature, rapid pulse-rate and delirium she was plainly a case of severe toxemia. An enema was given every hour and a half, and at 6 o'clock the next morning she passed a very large amount of foul-smelling, putrescent fecal material. From that time on her condition rapidly improved, and at the end of six days she was placed on a weighed diet, balanced with insulin. She was admitted on September 13. By October 13 the diet with insulin had been raised from 420 calories per day to 1470, and decreasing doses of insulin were required. One month after admission to hospital the child was taken off insulin and placed on the same diet as she had six days after admission, namely, 420 calories, and that diet was gradually increased until her discharge from the hospital on November 14. She remained sugar-free on that increased diet and without insulin during the

second month. Her diet was increased to 1470 calories and contained over 100 grams of carbohydrate. This is an example of coma precipitated by severe toxemia where the tolerance is temporarily overcome, and in such an emergency insulin is of very great value, and its administration is accompanied by a relief from overstrain and an increased tolerance.

The symptoms of diabetes which are primarily due to the failure on the part of the body to burn carbohydrate give rise to accumulation of sugar in the blood and the appearance of sugar in the urine. Since the tissue cells cannot burn sugar there is a continual demand for food and at the same time progressive loss of weight and strength. Also, since patients take so much fluid they pass large quantities of water. When insulin is administered in adequate amount it enables even the severe diabetic to burn sugar and thereby relieves all the cardinal symptoms.

I would like to express a word of appreciation to my associates: To Prof. MacLeod, whose direction has contributed so largely to the success of physiological development; to Prof. Henderson, of the department of pharmacology, who has enabled me to continue work at Toronto University; to Dr. Graham and Dr. Fitzgerald, and especially to my first associate, Mr. Charles Herbert Best, by whose efforts the large-scale production has been rapidly accomplished. I also wish to express my appreciation of the very admirable work done by the Lilly Research Laboratory, under the directorship of Dr. Clowes, and to all of those diabetic clinics of the United States who have coöperated so wholeheartedly in making insulin available for general use, and who have done so much to contribute to our knowledge of the disease, in order that treatment would be safe for the diabetic. But, above all, I wish to express our gratitude to the diabetic patients themselves, who are so appreciative of the benefits resulting from the treatment.

NEUROLOGY: PAST AND PRESENT[1]

By JOSEPH COLLINS, M.D.
NEW YORK CITY

The generation of man now at its apogee has witnessed many marvellous changes: It has seen the ultimate constituents of matter resolved into units of positive and of negative electricity, protons and electrons, and it has conceived the whole diversity of the world as due to varying combinations of them; it has seen the ether of the earth begin to yield its secrets and throw off its disdain; it has seen the dawn breaking on a diseaseless world and the recognition of the rights of man to live healthily and happily; it has heard the slogan, Peace on Earth, Good-will toward Men, which has heartened us for two thousand years, denied and destroyed. Countless epochal events have dropped the scales from our eyes and overcome the blindness imposed by ignorance and superstition. One of the results is the human body has regained some of the prestige it had in the days of Praxiteles. We are neither ashamed of it in repose nor embarrassed by it in action. Though it is not yet given the esteem and regard it deserves, no one viewing life retrospectively can doubt that the day of scorn of the flesh, contempt of the body, disdain of its normal cravings and denial of its legitimate desires is fast disappearing, and with its disappearance goes the monstrous delusion that the soul profits by studied contempt and pitiless humiliations of the body.

Puritanism, Philistinism, Potterism are by-products of this metamorphosis, and, like the by-products of metabolism, they are injurious if retained in the system.

[1] Read October 3, 1923.

The ills of the body are of three origins: design, accident, imposition. Under the first caption are the inherited and family diseases; their origin is still wrapped in mystery. Under the second are those due to infection, injury and intoxication; they constitute the majority quantitatively. Under the third, those due to fear and intemperance; the majority qualitatively.

The neurologist has to do with them all; but the first group he quickly hands over to fate, the second to the parasitologist, syphilologist and general practitioner, and with the third he labors. He does not preëmpt the field, nor, it must be admitted, has he tilled it satisfactorily. In this country, especially, it is constantly invaded by the supernaturalist and the legislator.

Despite the fact that neurology knocked loudly and clamorously at the door of general medicine, asking and demanding admission for a long time, its birth, development and reformation took place within three generations, from 1840 to 1920. In the first of these generations nervous diseases were looked upon as the farmer of bygone days looked upon black sheep; in the second, as he looked upon refractory members of the flock who were beyond correction; and in the third, as a prized portion of the flock, neglected but never disowned.

When I began to study neurology, physicians of this country had the same respect for neurology that tradition has for the owl. They held that it looked wise, but that was the confine of its virtue. It is remarkable how many of the conspicuous men in the profession at that time, especially the teachers, were from Missouri.

The physicians who believed it was an integral and important part of general medicine were active and articulate, and at the end of their generation they had convinced their fellow-practitioners that many diseases of the nervous system belonged to them. They delivered tabes, spastic spinal paralysis, paresis and some varieties of epilepsy over to the syphilologist and told him they were invariably the neural manifestations of the disease that he was pledged to recognize and treat, and intimated that it should be the training of every syphilologist to detect such

diseases in their incipiency. They told the ophthalmologist, and harped upon it, that he had the vantage position of all disease detectives; that to him was frequently given unique opportunity to discover serious disease of the nervous system in its very earliest and curable stages. They warned the internist that his studied refusal to cultivate familiarity with the nervous system and its reactions would redound to his material and moral disadvantage, especially when the infectious diseases of the nervous system and its covering would be handed over to him. They hinted that there was reason to believe that many nervous diseases, functional and organic, were the result of disorder and disease of portions of the body that the general practitioner considered his domain—for instance, the organs that subserve digestion and metabolism—and that it is as necessary to correct such disorders if you would get rid of the nervous disease as it is to close the polluted springs that furnish the water that mediates typhoid fever. They sought the intimacy of the dentist and the laryngologist that the incubus of "sciatica" might be lifted from them and the nightmare of "neuritis" dispelled, and when they had secured a measure of their confidence they suggested that teeth and tonsils discriminatingly treated would cast out more diseases of the sympathetic nervous system than were recognized or even suspected, and that to treat disorder of a ductless gland as a disease of that organ alone was comparable to a man who attempts to make a room habitable in winter by replacing a broken window-pane while he leaves the door open.

The result of the appeals, the warnings, the suggestion of neurologists was that teachers and practitioners at the beginning of the present generation awakened to the fact that neurology was an important branch of general medicine. When plans and determinations were made to teach it and thus to familiarize the profession and its descendants with its manifestations, and when there were successfully developed centers and institutions in which nervous and mental diseases were studied and interpreted, the neurologist's days of wailing and lamentation were over.

The neurologist cleared the jungle and invited prospectors and

tenants to stake their claims. These claims are now being cultivated for the relief and benefit of humanity. Here and there corners of the jungle remain. We call them disseminated sclerosis, dystrophies and hysteria. But the neurologist, aided by the bacteriologist and biologist, is at work with axe and lever, cutting and uprooting. When the task is accomplished, will he be reduced to the despair of Alexander? There is no likelihood of it unless our present system of ethics is subject to reconstruction or radical modification.

That is where we stand today. Are our moral laws, our ethical standards, our social conventions inimical to the development and preservation of psychoneural integrity? The majority of the neuropsychoses and the devastating frictions and minor ills of life have their foundation in fear—in fear which flows from ignorance, superstition and false teaching. Man's dominant fears are from the material and from the spiritual standpoints that he will not go through life successfully; fear that he will fall ill, suffer and die before he can fulfil himself; fear lest death may mean eternal darkness. It is the duty of the physician to dispel the former fears; of the priest the latter.

No human being is justified in forcing upon another defenseless and virginal beliefs and convictions not founded in facts. No human being is justified in withholding from them who are dependent and beholden the truth about themselves and the world, their relationships and obligations.

More suffering and misery result from ignorance of the species-perpetuating endowment, commonly called the sexual function, than from drink, drugs and dissipation combined. Practically all the so-called functional, neuromental diseases are traceable to some humiliation, denial or maltreatment of the creative urge, its foreshadowing and early display.

Children must be told the truth about sex in the same way as they are beginning to be told the truth about history. They must be told it when they first become concerned with sex—that is, when they become sentient organisms. They cannot take in the whole truth then; it must be modified as their milk is, but it

must contain the necessary number of emotional and psychical calories, and it must be palatable and assimilable.

Who shall teach them? Who knows the truth? Has it ever been formulated? Would not formulation of it be opposed to the principles of Christian ethics? Who knows the truth about history, the false teachings of which are held by many to be the chief cause of world wars? How are nations going about it to purge their text-books of false statements and frame those consistent with facts acceptable to the whole world? Physicians should know the part that aberrations, inversions, humiliations of the genesic instinct and perversions of its conspicuous function play in the causation of unhappiness, inefficiency, disease. But do they know it? Does not truth compel us to admit that the average physician knows no more about it than does the average layman? The neurologist knows, and it is his duty and privilege to share this knowledge with his fellow-practitioner and urge him to coöperate in the instruction of actual and potential parents, pedagogues and priests.

What does the parent solicitous for the spiritual and physical welfare of his offspring, determined to do everything that he can for its benefit, do now? He tells his child that it is injurious to masturbate and sinful to conjugate. He does not know that either statement is true. He has heard or read the former and his spiritual adviser has told him the latter. He knows, in all probability, that as a child and youth he sought and obtained vicariously or unnaturally appeasement of sexual desire, and he considers himself one of those who was fortunate enough to be absolved from paying the penalty.

He understands the seventh commandment to apply to the joined and the unjoined, and he would that others should obey it. He has no intimation that a considerable proportion of human beings come into the world with partially or completely inverted genesic instinct, and that the sanest and wisest minds of the past three thousand years have agreed that measures should be adopted to direct this endowment toward the heterosexual goal. It has never occurred to him that the plant, the mechanism, the opera-

tion of the genesic instinct is one of the profound complexities of Nature, and that its subjugation and artificialization has been one of man's strifes and pleasures for countless generations. He does not tell his child that there has been vouchsafed to him the most wondrous possession of the world—the capacity to create human species. He does not point out to him that the prelude to such creation is the most ennobling and soul-stirring event that the human organism can experience; that the act itself links man with God and fulfils His command. He does not reason with his child to convince him that a sensible and sane person does not drag his most treasured possession in the mud or wantonly tarnish his chief ornament; does not insult, outrage and maim his most faithful and valiant aid; and ruthlessly throw to the dogs the treasure that he has inherited and which he was created to conserve and perpetuate. He does not seek to convince him that it has been the consensus of opinion that wise men who have lived during those centuries in which such a progress that transcends understanding has been made in every field of activity, that such contact with the opposite sex should be under the seal of matrimony and that the family is the institution of which the world is proudest and which will last the longest. He spends infinite pains and money to teach him to dance, play games, to prepare him for college; but he considers that a few minutes' talk, in which he warns him of the "nastiness" of masturbation and the danger of sexual intercourse, is sufficient to orient him in the chief problem of life. He himself has realized that the aim of life is happiness and that the way to ensure it is to fulfil one's somatic and spiritual destiny and help others to do the same. That is, I take it to be what Christ meant when He said that "Love your neighbor as yourself is the greatest of all commandments." Despite this knowledge, he studiously neglects to instil into his child's mind the facts which, with health, are the pediments of happiness.

We take the greatest pains to select our Supreme Court from the wisest and justest lawyers of the land. They interpret the law, they pronounce its constitutionality. We spend countless millions framing, interpreting and administering such law to

protect our person and our property. We have no law-makers or interpreters of our personality and our spiritual property. Should anyone say we have the Church, I would ask him what instruction in biology and psychology its servants receive and what they offer save the word of the Master and of Paul? Does anyone maintain that the world could be administered today on the rules or laws laid down by them? Why should we expect that the spirit could be thus administered. Is evolution only of the body and not of the spirit as well?

There should be committees in every state drafting plans for the best way to teach children about sex: what to tell them and how; what to show them and when; what means should be utilized to keep presenting the complexity of the subject to the childish mind as it goes from bud into flower. These plans should be submitted to a supreme court made up of psychologists, biologists, priests, pedagogues and philanthropists for interpretation, simplification, elaboration and for applicability. Children should be taught the truth about sex with the same thoroughness as they are taught the multiplication table, and some means must be devised for early detection of those who have the misfortune to be born into the world with inadequate, perverted, inverted and excessive genesic endowment. They must be picked out with more care and discrimination than are the "atypicals," constituting the "unclassified" of our public schools.

The truth must be told about the effect on the mind and the body of sexual indulgence. The physician must speak from the point of view of one pledged to safeguard his fellows' health. If it is his conception of his part to speak from the moral standpoint as well, then he may do so.

The problem is a tremendous one, but no problem is ever solved, save by accident, until it is fairly and squarely stated. This problem is infinitely of vaster importance to the world than the malarial problem was when Ross attacked it twenty-five years ago, after Laveran, nearly a generation before, had shown it to be dependent upon a parasite, and after Manson had suggested that the mosquito was the bearer of the parasite to man, for it

concerns the whole world. We cannot expect the state to solve it until private and unselfish interest shows that it is solvable.

If we are to curb inadequacy, overcome inefficiency, thwart misery and promote happiness we must teach children that their sex endowment is their proudest possession. Parents must charge themselves to safeguard it so long as they are dependents, and, as an earnest of the light that has been vouchsafed them, they must counsel their progeny when they come to the age of reason how such possession may be enhanced and retained.

I purposely refrain from attempting to state specifically what physicians shall say to parents and progeny about sex continency and indulgence. There must first be agreement as to what shall be said, and before such agreement can be reached there must be discussion. It is for such discussion I plead.

The physicians of this country of the past fifty years have been strangely unmoved and pathetically untutored by the development and display of one of the spiritual wonders of the age—the birth and growth of Christian Science. It combats fear of disease. The person who does not admit that it is successful is as bigoted and beyond reason as he who does not admit that the tenets of its doctrine are inimical to science. Here is a system of therapeutics that has cured thousands and aided millions. "The mind governs the body, not partially, but entirely." This conviction is the most efficient agent of medical practice.

> "When the fight begins within himself,
> A man's worth something."

Physicians know, though Mrs. Eddy denied it, that diseases, death, suffering, sin, poverty and matter are realities. Knowing that, and having it constantly before their eyes that Christian Science often cures disease that medical science fails to cure, it is beyond explanation that the latter has not borrowed from the former, appropriated its truth and abjured its error. My observation is that the medical profession has not made the gesture, though here and there it has attempted to do so, as in the so-called Emmanuel movement. Such procedure is bound to be comparatively unsuccessful, for it dichotomises the therapeutic nucleus,

one portion going to revive the sick soul and the other the ailing body; whereas the infirmity of the latter is dependent upon the inadequacy of the former, and it could be readily overcome were it detected. To doctors their fellows go when ill, for they are the detectives of disease.

I plead for the recognition of their illness, imaginary or real, spiritual or somatic, and for its appropriate treatment. It would be futile to expect that physicians who had not been properly trained could do one or the other save by happy accident or intuition. When it is recognized that the mind plays such a leading role in the comedy of life as Quimby and Coue, Eddy and Freud, Mesmer and Perkins, and countless other supernaturalists and neoplatonists have shown, then the forces that shape the policy of our medical schools will realize that it is as important to teach psychology as it is to teach physiology.

The remedy for aberrational by-products in pseudomedicine, pseudopsychiatry, pseudopsychology and pseudoethics is to focus the search-light of correct and adequate knowledge on the functional equipment of behavior and the problems that such equipment pose. When psychology is an integral part of the curriculum for medical students, and when physicians give the same close attention to the study and treatment of diseases of the spirit that they do to diseases of the body, faith cures and miracle cures will become less, for their source of supply will be cut off.

My final plea is that the mantle of secrecy which the physicians and family of those so unfortunate as to develop mental diseases throw over them should be vigorously dragged away and that the infirmity should be dealt with in the open, as it were. An individual should be no more ashamed of mental disease than he is of physical disease: in fact, he should be less so, because he is less responsible for it. Our present attitude toward it is a relic of the days when it was considered a demoniacal possession.

Most mental diseases do not render the victim insane in the conventional or in the legal sense of the term. Not one psychopathic individual in twenty needs to be deprived of his liberty in

order that his welfare may be studied and that of the public safeguarded. Such mental diseases are habitually neglected by physicians. They are compelled in the vast majority of instances to take their grist to the supernaturalist miller. He promises to winnow their grain and to grind it. He learns how to do it by experience.

Personality disorders should be studied and treated in general hospitals and dispensaries by physicians whose temperament and training qualify them for psychotherapy. If these physicians studiously immunize themselves to the leaven that has been made available by rest cures and work cures, suggestion and autosuggestion, psychoanalysis and psychosynthesis, "New Thought" and "Old Thought," they put a handicap upon themselves to the disadvantage of the patient and to the discredit of their art.

Psychotherapy should include every form of treatment that has been shown to be useful by man or superman, physician or priest, healer or manipulator. The privilege of this physician is to show how such cures conform to physiopsychological laws. Mental disorder or disease displays itself in personality alteration. Until the training of physicians, compulsory and voluntary, includes study of character we cannot hope to see applied psychology make considerable progress.

This takes us into the field of education, a field that is now being tilled by implements and methods that are as archaic as the mattock and plough of Virgil. The object of education is to fit one for the business of life. Its opportunity is to supervise the habit-forming activities of the young. To this task the judicially appraising, scientifically scrutinizing work of the neurologist can contribute to correct the fads and fancies of the emotionalist and the delusions and obsessions of the fanatic.

The physician of the future, especially the neurologist, must contend with intellectualism as the priest contends with materialism. They are the evils of the age. Concentration on intellectual development must give way to character-building, and training of the memory, "Memory the warder of the brain," to training of the reason.

The problem of the psychologist is to estimate and administer

the power of the mind; it contains forces which have not been fathomed, possibly not even suspected.

There is a new way of regarding the human body and there is a new way of regarding its ills. The neurologist has contributed to both; directly by what he has added to our knowledge, and indirectly by his influence on psychiatry, psychology, medical and social service. It is selfish of him not to share his possessions with other members of his guild and timorous of them not to demand it.

DISCUSSION

Dr. Charles K. Mills: In the discussion of this paper we can only say that Dr. Collins has put before us some ideas which in their fruition can do much, not only for the neurologists but for the individuals with whom the neurologists have to deal. I endorse much of what he says without being in a position to thoroughly discuss it.

Dr. F. X. Dercum: In listening to this paper I have been impressed especially by the high ethical standard and point of view with which the subject has been approached. In ancient times the priest and the medical profession were one—the priest was the doctor and the doctor was the priest—and it seems to me that the paper as read is one to arouse in us all a realization of our full responsibilities. Every doctor's office is in a sense a confessional and there are indeed very few physicians who do not, in a measure, discharge both functions. We are so often called on for ethical advice, not necessarily medical in character, but dealing with the very problems which Dr. Collins has suggested in such beautiful language. To me the paper is appealing as indicating that we should fulfil in the future, even more than in the past, this double function.

Dr. Daniel J. McCarthy: I think all neurologists agree with what Dr. Collins has said, and especially to what he says about the study of the cases and the teaching of psychology in the medical schools.

The duty of the doctor is to stop and investigate the case in front of him and find out what are the factors that enter into the life of the patient. I think the teachings of Freud have had much to do with our appreciation of the necessity for taking complete histories. I doubt if before Freud's time any of us would have thought of taking 20, 30, 40 or even 50 pages of history for one patient. I feel that the future of medicine is the time we spend in the study of the individual cases. I think we should teach students psychology.

Along these lines I would like to describe an incident which occurred in my office recently: Two resident physicians came to me for advice on an ethical question. A bootlegger apothecary had come to them with the proposition that he would give them a large amount of money if they would let him use their names on prescriptions for alcohol. Both men were about to leave the hospital and start in practice, and both needed money very badly. I told them they should certainly not want to sell their birthright for a mess of pottage, but they resented this attitude and said they needed the money to practice, and where were they going to get it. I told them as they only were busy in the hospital six or eight hours a day they should certainly be able to get some work at night, and suggested that they give massage to some of my patients in the evening. They replied that they did not know anything about massage. This I think is amazing and remarkable, and I have offered to give them some instruction in the fundamental principles of massage. It seems incredible that two resident physicians should know nothing of the principles of massage. I remember when I first started in practice I did not hesitate to give massage myself to the patients when they required it. I think physicians should be able to give their patients something more than a prescription if they need it. And there is no reason why young physicians should be starving when osteopaths and masseuses are making money. I think the time has come when we should go about plans for having students taught something more than the mere writing of prescriptions. In dealing with the families, and especially in dealing with children, we are dealing with fine spun gold. I believe that the child's physical care is most important, the removal of diseased tonsils, etc., and I think a sound, complete body coming to adolescence in a normal life with normal environment will come through without much difficulty. Of course, we must never lose sight of the pathologic personalities.

Dr. Owen Copp: I am much interested and indebted to Dr. Collins for his beautiful presentation of an important subject and have one thought to express, that this subject should come into the thought of every physician in hospital work. That means such a service in connection with every general hospital, completely equipped for the study and practice of general and special medicine.

Dr. Collins (closing): Bearing on Dr. McCarthy's remarks, I should like to call your attention to an article in the *Illinois Medical Journal*, a few weeks ago, entitled, "The Laity's Idea of the Physician." The subject has only an indirect bearing upon my paper this evening, but between the lines of my address I hope to convey that there is a hiatus between the doctor and the laity which should be obliterated.

Dr. James H. Hutton, of the Chicago Medical Society, suggested and financed an inquiry why the people of Chicago patronized quacks in

preference to doctors, supernaturalists in preference to trained physicians. He interviewed about 7000 persons; all classes were represented, from day laborers to society leaders, with just about a 50–50 break in figures between those above and those below the middle class. Of this number 5841 were directly against the physician and equally directly in favor of the supernaturalist. Some of the replies that they gave to the question, "When you were last ill, why did you go to a practitioner of naturopathy, electronic reactions, soul-science, medicoradio vibrations, to an osteopath or Christian Scientist and not to a regular doctor?" are amusing, others are imbecile, but all of them show profound dissatisfaction with the physician. They may be responsible for their belief, not the doctor, but one cannot help feeling that if the latter would drop some of his side, aloofness and, may I say, air of superiority and infallibility and discuss medicine, its possibilities, limitations, probabilities and certainties, frankly and openly, that much of this distrust and loss of confidence would be eliminated. I am sure that it is in some such way that we must discuss the creative function of man, openly and frankly.

We must tear away the shroud of shame in which the sex function has been wrapped. We must tell our fellow-men, when they are as yet miniatures of men, that they should be proud of their sex and not ashamed of it; we must bury the masturbation-bogy and whatever our feelings may be concerning deviation of the genesic instinct; it is our duty to look upon it as an aberration of Nature, not a vice, and do what we can to get the genesic current back between the banks that lead to the heterosexual ocean.

You may ask, "Are neuropsychiatrists agreed on what you have stated, and, if so, why do they not undertake the educational propaganda that you suggest?" and in reply I should say, "I think there is as much unanimity of belief in this matter as it is well for a group of men and women to have upon any matter." It would be futile for them to attempt to carry on the propaganda. It is the general practitioner who has both the opportunity and the privilege to do it. The neuropsychiatrist and the general practitioner must get together on this matter and they must have a third member, one whose concern is the spiritual welfare of the individual. These three must decide the truth about sex. Now if the general practitioner does not wish to sit in on such committee he can be replaced by a pedagogue, and after they have deliberated and decided they can hand their suggestions and commands to the general practitioner for execution. I need scarcely say to you, in closing, that I have a keen realization of the difficulties that will be encountered in carrying out such a scheme, but we cannot go on treating sex in the Paulian manner: such treatment is responsible for too much suffering in the world to allow it to continue.

THE SOCIAL SIGNIFICANCE OF MEDICINE[1]

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In times of peace the individual is prone to an exaggerated assertion of self at the expense of society; in times of war, or when war is threatened, bonds that hold individuals together in a nation become firmer and collective interests temporarily become paramount. At all times it is desirable that a proper balance be maintained between the activities that favor individual or group welfare and those that favor the welfare of society as a whole; for, on the one hand, the individual must do well if society is to prosper, and, on the other hand, society must be preserved and strengthened through the loyalty, devotion and obedience of its units, even at the sacrifice of some of their individual interests, if the individual is to have freedom of opportunity for the exercise of his faculties. It would seem desirable, therefore, that each person, and each occupational or professional group, should, occasionally, give some special thought to the matter of his and its social meaning and value. This is my excuse for asking your attention, tonight, to a brief consideration of the social significance of medicine.

In estimating the value of a person or of a professional group to society, one must try to determine the relation of the activities of that person, or of that group, to the attainment of the aims of society as a whole. In our modern civilization the aim of society would seem to be the attainment and maintenance of those conditions in its members and in their environment that will, to the greatest number of units, permit of the greatest freedom in exer-

[1] Read October 3, 1923.

cising harmoniously their various faculties, each for his own and at the same time for the collective welfare. Theoretically, in a perfect state, the men and women who constitute it could and would lead lives through which the highest individual good and the highest collective good would simultaneously be realized. But the perfect state never has existed, and never will exist, except as a fiction, an ideal toward which we may work, a standard by which our imperfect collective achievements may be measured and valued.

If these preliminary assumptions be agreed to my task of this evening resolves itself into a consideration of the importance of the functions of members of the medical profession for promoting the freedom of social units to live full and well-proportioned lives, lives that are individually good because of proper exercise of all the liver's faculties and that promote the collective good because in them the social virtues are practised. And since the kind of life a person lives depends upon his inborn capacities and upon the surroundings these are immersed in after birth, the business before us is to inquire in how far medicine does and can influence the constitutional and environmental conditions of man, which determine his functions and the modes of their exercise. In undertaking as its province the maintenance of health and the prevention and cure of disease in human beings, the medical profession has entered upon a gigantic enterprise, one that makes great demands upon the heads and hearts of those who engage in it, one that is full of promise for the welfare of individual persons, and one that when comprehensively viewed will be seen, I must believe, to be so significant for the whole of society as to demand, indeed to guarantee, society's warm support and high esteem.

If the view of the functions of medicine that I shall try to outline be well-founded, then our profession can lay claim to at least equal rank with her sister profession of the law in importance for the promotion of collective welfare. The great philosopher David Hume, speaking of the law, said: "Man, born in a family, is compelled to maintain society from necessity, from natural inclination, and from habit. The same creature, in his farther progress, is

engaged to establish political society, in order to administer justice, without which there can be no peace among them, nor safety, nor mutual intercourse. We are therefore to look upon all the vast apparatus of our government as having ultimately no other object or purpose but the distribution of justice, or, in other words, the support of the . . . judges. Kings and parliaments, fleets and armies, officers of the court and revenue, ambassadors, ministers, and privy councillors, are all subordinate in the end to this part of administration." Truly a noble view of the law. My aim is to show that medicine, which has as its object the healthy mind in the healthy body, and which devises and executes eugenic and euthenic programs directed toward this end, performs a service to society that is equally indispensable, a service, indeed, upon which, on due consideration, the very practice of justice itself will be seen to depend.

Medical men in ancient times doubtless began their activities by striving to be of help to those who were sick, injured or otherwise in distress, applying all the simple remedies that the method of trial and error taught them to be of value for their care or alleviation. From these primitive beginnings the great work of the medical profession has gradually evolved. The curiosity excited by deformity and disease stimulated the simpler observational investigations that were the forerunners of the abstruse researches of the modern medical experimenter and discoverer; the primitive remedial activities became gradually extended to culminate in the rich therapeutic armamentarium and the elaborate practical-technical methods of the present-day medical and surgical practitioner. The recognition of the disastrous consequences of disease, stimulating observation and inquiry into conditions of health, gave rise to the earlier rules for the preservation of health and the prevention of disease that have been transformed during centuries of change into the more precise and further-reaching regulations of contemporary personal, communal and racial hygiene. And the methods of passing on medical lore from person to person, from experienced master to apprenticed pupil, gradually underwent development and differentiation into our modern agencies and

institutions for the diffusion of knowledge of medical science and for training in the art of applying it—our great schools of medicine and of hygiene with their specialized faculty members, our libraries with their rich stores in medical text-books, monographs and journals, and our local, regional, national and international medical associations for the quick transmission and wholesome, penetrative criticism of new medical facts and impulses. Out of the chaos of ancient medical myths the whimsical and extravagant notions of antique therapy, the hygienic traditions of primitive peoples and the defective early methods of transmitting medical knowledge have emerged the medical research, the medical practice, the preventive medicine, the public health measures and the scientific medical education that are characteristic of the civilization of the twentieth century. Those members of society to whom have been entrusted the special function of preserving health and of curing and preventing disease have undoubtedly through the ages felt in ever-increasing measure the magnitude and the seriousness of their responsibilities. They have achieved results and left records that give abundant reason for professional pride; they have opened paths for work that are full of promise to those who will further walk in them, and they have by their intellects and imaginations conceived of worthy ideals that spur us, their successors, to devote our best efforts to the attempt to realize them.

Since society achieves its own welfare through the life-activities of its constituent units, let us examine the ways in which the medical profession of today becomes socially significant through promotion of the self-realization of individuals in social service. What does the medical profession do, on the one hand, to help equalize individuals in their freedom to exercise all their functions, to find adequate expression for the whole of their personality, and, on the other hand, to aid social justice by favoring a balanced proportion among the interests of those who have equal rights to the possession of interests? In other words, in how far are physicians of use in helping people to direct their own lives by reason and conscience so that they will, while exercising their own powers to the full, perform their social functions better; and in how far are

they influential in helping persons not only to defend the right to be themselves but also to recognize the obligation to refrain from interference with the rights of others? The greater the service medicine can render to society in these two main directions—that of facilitating the free and harmonious exercise of their functions by the social units and that of harmonizing the activities of these units so that in society there may be fairness for all—the larger will be its social import.

Medical investigators in their studies of physical diseases and of abnormal thought, feeling and behavior have discovered and described enormous numbers of physical and mental maladies and irregularities. They have, too, more or less successfully inquired into their causes, and they are ever striving to follow in series the links in the chains that connect these causes with those ultimate consequences that we know as signs of structural change and as symptoms of pathological functioning. The causes that they find are sometimes defects in the original makeup of hereditary qualities, sometimes malign influences—physical, chemical, biological or social—in the environment and more often still a combination of faulty hereditary tendency with environmental injury. And among the consequences of abnormal function it is always interesting to observe in how far the damage to the individual and the corresponding hindrance to his welfare impair also his social usefulness, or, as they do in many instances, make him actually a social menace. We know that physical disease involves both personal and public loss. Malaria and yellow fever not only prostrated their victims, but they prevented for a long time the development of Cuba and the building of the Panama Canal. The tubercle bacillus, the *Treponema pallidum* and the hookworm render miserable those who are personally attacked by them; they also are an economic drain and they exert a dispiriting influence upon the families of the infected. Similarly, mental diseases and abnormalities are subversive of both personal and collective good; the insane, the imbecile, the criminal and even the psychoneurotic lead very imperfect lives themselves; but, besides, they lessen the happiness and increase the expenses of the families they belong

to and they often seriously endanger general welfare in the communities in which they live. It is to the credit of modern neurology and psychiatry that they have done much to further social progress by changing our attitude toward social offenders. In the perpetrators of social evil they have sought the true causes—physiological, psychological, economic, social—of their moral obliquity and are pointing the way to remedies that those flooded with the passion of punitive hate could scarcely find. Medicine, long ago discovering the rôle of natural law in disease, rescued the physically sick from those who would exorcise demons; later, she redeemed the insane from the cruelty of the moralistic judgment, the fetters unbound by Pinel remaining, let us hope, permanently unloosed; now, through the light shed by psychiatry, she is extending her scientific view so as to include the criminal, the vagrant and the perverse, and will doubtless advantageously influence penology by gradual rationalization of the code of condemnation. Indeed, the rescue of "sinners" of various types seems to fall within the province of the physician, greatly enlarging the territory in which his functions find exercise. But in rescuing such "sinners," medicine does not approve of their unnecessary multiplication. Medical men, observing among social units, along with the fit, the many that are "notoriously unfit," will be among the most diligent of those who educate to social action that will gradually lessen the numbers of the latter.

The results of medical inquiries do not indicate, however, that it is desirable to have a society composed of individuals who are all alike, identical copies of an original pattern, no matter how perfect that pattern might be. For what could be more depressing than the thought of a multitude of human beings with precisely the same natures, possessing the same hereditary qualities in exactly the same degree, all reacting to environmental influences in one stereotyped manner? The very idea is enough to excite horror. No; for the attainment of the highest good in human society diversity rather than identity of form and function is necessary, and for this variety Mendelian heredity fortunately provides. Nor can medicine ever hope definitely to describe

every possible combination of germ plasms that will result in native qualities that will ensure physical and social fitness. It has, however, already discovered laws of inheritance that account for at least many instances of physical disease and deformity, and for some of the flagrant examples of social maladjustment and pathological behavior. It has shown us very clearly that society, if it is greatly to improve, must give more serious consideration than heretofore to the constitutions of its members as well as to the conditions under which they live.

Medicine, after calling to her aid many auxiliary sciences (biology, psychology, anthropology, sociology), and after prolonged and painstaking research herself, has learned much of the native dispositions common to all men and of their significance for human life. Among these native human inclinations may be mentioned the desire to be physically healthy; to protect and preserve the self and to propagate the kind; the desire to possess; the desire to associate with others; the desire to use the intellect; the desire to create and to enjoy beauty; and the desire for personal conduct that will satisfy conscience. In order that the individual may make the most of life for himself and that he may best serve society, his environment (including his education) should be such as to call forth reactions that will satisfy, in a harmonious and well-balanced way, each and all of these fundamental human needs. This does nothing to preclude variety in occupation or in recreation, for in no two individuals are the native dispositions existent in the same order or degree; moreover, in their environment, no two persons are subjected to identical conditions. The possible permutations and combinations are infinite and permit accordingly of infinite variety in both human nature and human conduct. For every human being is a unique person; there is and never will be another just like him. This, though a platitude, has ever to be reëmphasized.

One of the most important of medical tasks, one that is socially highly significant, is that of advising persons regarding hygienic living. But, it may be asked, how, if no two human beings are alike—and it is desirable for the good of society that they should

be unlike—can rules of living that are applicable to all be valid? Must not every man have rules that pertain to him alone? Must he not find, for himself, what diet, what exercise, what work, what association, what recreation and what conduct are best for him? To a certain extent this is true. There can, perhaps, be no universal dietetic-hygienic régime in the concrete. A yankee in New England does not eat nor need the same diet as the darkey in Mississippi, nor are their actions in other respects identical. What is suitable and essential in habits and morals to the one need not be so to the other.[1] But despite the diversity in human beings, there are such similarities that it is possible, as we all know, to outline certain rules that are, in general, valid conditions for the preservation of bodily health and also certain rules that are, in general, valid conditions for the leading of a socially useful and a spiritually elevated life. In this limited sense, medicine can establish a universal physical and mental hygiene, even though the hygienic adviser in attempting to apply these universal rules to individual lives must know how to adapt them to each person's particular constitution and condition. The hygienic adviser should never forget the uniqueness of each human being. Because of the uniqueness of his hereditary qualities and of his surrounding influences, each person actually requires a special bodily hygiene, special rules for thinking and enjoying, and, yes, even a special code of morality. This is true of what we call normal persons; it is, moreover, increasingly true of the physically diseased, of the mentally disturbed, of the socially maladjusted, of the morally oblique and of those who are deficient in feeling for beauty.

Who, then, has a larger or a more responsible function than he who undertakes, as the physician is now compelled to do, to act as personal counsellor to people in the conduct of their lives? How extensive should be his knowledge and experience, how great should be his power of imagining himself completely in the place of the person he advises—what has been spoken of as the power "to creep under men's skins" and with what infinite tact must he

[1] Cf. Paulsen, F.: A System of Ethics, English translation, by F. Thilly, New York, 1908.

be endowed if he is to be able to convince and to persuade! Can there be any doubt of the social significance of the performance of such functions, of the value to society of the performers if they do their work well?

It is upon the general practitioner of medicine, aided by the consultant and the specialist, that the bulk of this work of advising persons of diverse character, and in the most different conditions, as to hygienic living, falls. It is to him that the people turn for counsel, especially when disease exists or threatens, but nowadays more and more also, in order that the bodily health and the capacity to work may be preserved, that mental functions may be exercised freely and that conduct may be conducive to personal and public welfare. He it is who watches the digestive, the circulatory, the respiratory, the hematopoietic, the urogenital, the locomotor, the nervous, the endocrine and the metabolic functions, and determines when therapeutic intervention is necessary. He educates his patients to avoid everything that impairs these functions and to do everything that will strengthen and harmonize them. He warns when necessary against caprice in eating, against undereating, against gluttony, against the misuse of alcohol or tobacco. He teaches the importance of a balanced food ration of appropriate caloric content, the necessity of considering health as well as fashion in clothing, and the value for health, happiness and efficiency of a well-arranged program of work, of leisure and of recreation. He tries to stimulate the idler to fruitful industry, the spendthrift to frugality and the miser to liberality, for idleness, extravagance and avarice are vices that are both injurious to the individual and harmful to social life. To those who have secured incomes that more than suffice for their biological needs, he points out the dangers of hypertrophy of the possessive instincts and encourages the promotion of the intellectual, social, esthetic and ethical interests. Indeed, the physician can often be most helpful to his patients in guiding them to sources of truth, to desirable companionship, to participation in community efforts, to the enjoyment of the beautiful in art and literature and to worthy conduct that conforms to more ideal standards. There are those

who maintain that the domain of the physician does not extend beyond the body of the patient and its perfection. I cannot agree with them, but were we to accept their view it might still be urged that beauty and grace in the body can be attained only through proper cultivation of the mind and spirit. Self-mastery is essential to bodily grace, and only when the soul is good and beautiful can the highest type of bodily beauty be realized. Moreover, in order that health of mind and body may be maintained, it is essential that the whole personality of the patient be developed in an all-round and proportionate manner. Undue suppression of the sex-interests, of the possessive interests, of the intellectual interests, of the social interests, of the interest in the creation and appreciation of the beautiful, or of the interests in right living, will react unfavorably upon either bodily or mental health, or both. Thus attention to all these interests must obviously fall within the province of the physician, and he who conscientiously strives to perform his duties to his patients does not neglect them. In lending his aid, however, to his patient in attaining to the realization of his total self, the physician will avoid making him too subject to his counsellor. The aim of the best hygienic educator will always be to lead his pupil to self-reliance and to independent self-determination.

Thus far I have referred chiefly to the social service rendered by medical investigators and medical practitioners. If time permitted, it would be interesting to review also the meaning and value to society of the activities of medical authors, medical teachers, medical organizers and medical executives, including the directors of hospitals, sanitaria and asylums and public health officers. But I must have some regard for the limits of your patience.

I cannot, however, forbear some comment upon the importance to society of the activities of certain members of our profession who may be designated, perhaps, medical statesmen. By these I mean those medical men of large vision who, better than their fellows, see the importance of the promotion of the science and art of medicine for the progress of civilization, who devise new

plans for improving the conditions of practice teaching and research, and who are helpful in passing better laws and in establishing better institutions, educational and political, for rendering the state-needed work of physicians and scientists more fruitful and more secure. These men are never ignobly content with the past or with contemporary achievements of the profession. They are the shakers of creeds, the questioners of dogmas and the critical surveyors of received traditions. They see ever new goals that should be sought, and they confront us with new problems—physical, mental, educational, industrial, social, international—that medicine can help to solve. They stimulate our interest and the interest of intelligent laymen in these projects; they prepare the plans for the work, and they find the men and the money necessary for these new ventures. Here in Philadelphia you had such a man, for example, in the late William Pepper; Boston has had its President Eliot; and in Baltimore we have had Professor Welch. Such men deserve, as did John Hunter and Lord Lister in England, Virchow in Germany, Billroth in Austria and Pasteur in France, to be called philosophical medical statesmen. That these men of clear insight, of vivid imagination, of ardent enthusiasm and of strong will may have a worthy line of successors should be the hope of all who cherish social good.

And, finally, the examples in living set by medical men themselves can scarcely be without their value for society. Though they have their faults and many of the common human failings, medical men, through their knowledge and training, should be better able than the average man to order their lives well, and, on the whole, I believe that they do. When they take care of their own health, marry happily, train their children sensibly, attain to economic independence, attend to their social duties, keep their intellectual interests alive, show their appreciation of the beautiful things in Nature and in the work of man, and respect the rights of others, they become artists in life and their mode of living is worthy of social esteem. For example is better than precept and lends force to precept. Those whose business it is to inculcate the principles of hygiene—physical, mental or moral—

will have a larger success if their own lives are not too discordant with their teachings. In the worthy satisfaction of his own fundamental needs, exhibiting restraint without undue restriction, and illustrating moderation that has no taint of meanness, each physician may strive to exemplify hygienic living in the larger sense and so further contribute to the social significance of his profession.

A distinguished Canadian jurist once remarked to me that any man might well be proud who belonged to a profession that deserved the high tribute paid to it by Oliver Wendell Holmes. The lines he referred to may be familiar to some of you, and perhaps I cannot do better than to close with them: "Charity," said he, "is the eminent virtue of the medical profession. Show me the garret or the cellar which its messengers do not penetrate; tell me of the pestilence which its heroes have not braved in their errands of mercy; name to me the young practitioner who is not ready to be the servant of servants in the cause of humanity, or the old one whose counsel is not ready for him in his perplexities, and I will expatiate upon the claims of a virtue which I am content to leave you to learn from those who have gone before you, and whose footprints you will find in the path to every haunt of stricken humanity."

DISCUSSION

Dr. James M. Anders: I think the subject of Dr. Barker's paper is distinctly timely and most important, and he has presented it in his usual masterful manner. What I have to say is intended not in the nature of criticism but as an addendum to what he has said better than I possibly could. He considered the subject from the standpoint of the individual and the medical profession as well as the general public. I have long felt, and have advocated in papers presented before medical societies, that physicians should establish and maintain a system of instruction in personal hygiene for the benefit of their patients, and now after hearing Dr. Collins' paper I think that personal instruction in psychology should be added as soon as the profession is ready to give it, which is not the case at present. While there are some general laws and facts in personal

hygiene which if followed out will be advantageous to the majority of patients, still we must always endeavor to adapt the instruction to the individual. I feel strongly that the custom of instructing patients in personal hygiene is not as general as it should be among the members of our profession.

In my view it would be well for the medical profession to undertake to teach the public all about the various cults, including Christian Science. I think the best way to do this would be to have an organization, preferably the American Medical Association, appoint a commission to investigate the various cults and report its results to the profession and to the general public, the object being to define as accurately as possible how much, or rather how little, virtue there is in each. My feeling is that there is some little virtue in all of these cults, but, of course, the claims made for them by their devotees are absurd. The medical man of today is a much more useful citizen, socially considered, than he has been at any previous stage of the history of medicine. One of the ways in which he has become an important social factor has been through his joining the various welfare organizations, such as public health associations, antituberculosis societies, village improvement societies, and the like, and owing to his special fitness for carrying out the aims and objects of such associations, he has done and is doing quite as much for social progress as he can, as an individual, dealing with his patients. The medical profession should try to put across to the public the fact that the progress and well-being of a municipality, state or nation depend largely upon its medical service and sanitary status or health rate. If this could be done successfully it would mean much for what Dr. Barker has so aptly referred to as the collective welfare."

Dr. R. Tait McKenzie: No one who sat here tonight could help being stirred by these two papers, taking up the question as they do from opposite points of view, the optimistic and the pessimistic.

I too was very much disturbed by the report of the dissatisfaction of people with the present condition of medical treatment, and it seems to me we are now at the time where the lines between the priest and the physician and the physician and the educator are rapidly shifting. A great many of the problems that come up to the physician are educational problems. The treatment of speech defects, faults of posture, ataxia and many heart conditions are all educational in character. In consequence of this we have to change many of our views on teaching in our institutions, so as to include this aspect of our therapy.

I was impressed with the vogue of the chiropractor this summer when I was lecturing in the University of California at Berkeley, to a class of

about 100 graduate students, one of whom was the president of a "college" and "clinic" which I subsequently visited and investigated. And, incidentally, let me say to Dr. McCarthy that every Monday and Wednesday at 5 P.M. we have a class on massage, corrective gymnastics and the application of electricity and hydrotherapy for the medical students of the second year at the University of Pennsylvania.

They are certainly increasing in number and influence largely through our neglect, and we have got to include the subject of physiotherapy in our educational program if we are to meet conditions as they are with success.

PREVENTIVE MEDICINE THE OPPORTUNITY OF THE PRIVATE PRACTITIONER[1]

By HAVEN EMERSON, M.D.
NEW YORK

(By Invitation)

We are, it seems to me, just entering upon the third and what I believe will prove to be the most successful and enduring stage of preventive medicine. The first era in the development of public health in this country extended from just prior to 1700, when the people of Boston demanded the quarantining of the crews and passengers on certain ships from Europe because of smallpox, until just prior to 1900, when the small beginnings of education in health were made by those interested in the prevention of tuberculosis. This era was marked chiefly by the control of sanitation and all the elements of man's physical environment through the authority of boards and officers of health acting under the police power of the states.

Great gains were made in the standard of living, in the disposal of human waste, in protection of water, milk and food supplies, and in the compulsory isolation of persons in the communicable stages of disease. This was the era of sanitary law, when the health officer was chiefly a glorified sanitary policeman, often out of touch with his community and the medical profession, and frequently more devoted to the abatement of nuisances that offended sight and smell than to the detection and removal of sources of infection in persons.

This era included the great triumphs over diseases spread through discharges from the intestinal tract and those transmitted by insects

[1] Read November 7, 1923.

and parasites. There was no great individual participation by the general public or by the medical profession in this accomplishment. The results in the main were attained by doing things for the public through official agencies.

The second era, beginning with the first antituberculosis committees in our larger cities just prior to 1900, has extended up to the present time, and its policies and results, its organization and efforts, will continue to be active and effective for many years to come; but the chief accomplishment of this period is completed, namely, the introduction of the element of personal education and information as to health and its attainment, disease and its prevention, life and its larger and happier development.

The typical expression of this era has been the development of the National Tuberculosis Association, springing out of the interest arising from the proof that the disease is communicable, preventable and curable. It can fairly be said that no effort of universal education as to the cause and means of prevention and relief has ever been made which reached so large a proportion of the people of any nation. This era has been marked more by the practice of education and voluntary effort than by enforcement of sanitary law alone. Coöperation and personal interest have been elicited, with the result that public opinion has run as fast as that of health officers and law became less important than learning. The sense of responsibility broadened from one man, the health officer, to large groups of interested and determined citizens, who expressed their public spirit in an effort to save lives through knowledge and adequate preventive and curative facilities.

Following the method and success of the tuberculosis pioneers came the agitation for prevention of infant mortality, the development of school medical inspection, the protection of the expectant mother, the supervision of the pre-school child, and at the same time there were created, to meet the growing conscience and demand of a newly aroused public, the American Social Hygiene Association to deal with venereal diseases and their prevention, the National Committee for Mental Hygiene, the American Society for the Control of Cancer, and lastly the Association for the

Prevention and Relief of Heart Disease, each one directing its chief attack upon the ignorance of the general public.

During this period of widespreading health education those responsible for each of the national organizations just mentioned soon appreciated that a single element was common to each effort, namely, the examination of the person and personality of each individual by a trained medical observer. It will be unnecessary before such an audience as this to detail the steps in arriving at this conclusion, but you will, I think, agree with me that each program of education in health leads logically and necessarily, if it is successful, to some specific thing to be done by the individual concerned. The action required of the person approached or appealed to by the educational work of each of these volunteer health agencies is the obtaining of a thorough competent medical examination by his family physician.

The third era developed inevitably out of this general recognition of the common objective of all the public and private health services of the country, and its origin was marked by two notable actions, the vote of the House of Delegates of the American Medical Association, in May, 1922, endorsing and advising periodic health examinations of presumably healthy persons, and the joint undertaking of the National Health Council, representing all the national health agencies, to urge and promote the demand for an annual examination of everyone for the purpose of early detection and prevention of disease, and to permit that relation of physician and patient which will put the former in the position of private health officer to each household in the land. All this means that we recognize the indispensable character of diagnosis for health as for disease.

We are now face to face with the logic of our accumulated experience, to the effect that, lacking a basis for personal conduct or hygienic manner of life coming from the advice of physicians, we can make little further advance in the reduction of sickness or postponement of death, and that with information based upon the skilled inquiry of a physician, we can closely approach the biblical ideal of longevity.

What shall be our plan of action as physicians and what our proper duty as individuals subject to the same ills as our fellows? What is the total program as it appears now from the point of view of the most important social and racial unit, the family?

If we think of life expectancy as approximately a term of sixty years from birth, we can with reasonable accuracy determine the number of occasions when preventive or health examinations should be made, to meet our present conception of the value and timeliness of personal service by the family physician.

On at least four occasions after the fourth month of pregnancy the expectant mother should have medical supervision, with the appropriate clinical and laboratory examinations, in the interest of the normal birth and survival of the child, as well as to secure the safety of the mother.

During the first two years of life there will be needed twenty opportunities for medical survey of the growing infant, a type of service commonly supplied by pediatricians, and to an increasing degree by general practitioners, either at special public baby stations or in their private offices.

From the third to the fifth year of life a medical examination should be arranged for every six months and oftener if there has been in the interval sickness and convalescence from any of the infectious fevers so common at this period of life.

From six to fifteen years a single annual examination should suffice.

From sixteen to thirty-five the minimum protection required may be given by a medical examination every two years.

After thirty-five the practice of an annual examination should be followed, and where there is any serious handicap, such as obesity or high blood-pressure, even if not causing obvious distress, the interval between examinations after the age of forty-five should be shortened to six months.

This means that on seventy-five occasions, during a life of sixty years, personal service for health protection is advised. I think such an investment will prove economical to any individual.

The difficulties in the way of this new program are, first, lack

of faith in the knowledge of the practitioner of medicine in the field of prevention, and, second, lack of skill, imagination and practice in the physiological and hygienic aspects of medicine by the physician himself. It is the burden of our medical schools, our hospitals and our medical societies to continue the education of physicians in the objectives, the method, the technic and the practical results of periodic health examinations. It is the function of the private and public health agencies to teach the laity to demand and obtain from physicians a service for health of as high a grade as that of the care provided in sickness.

Physicians cannot and should not advertise their personal ability to protect health or to cure the sick. The seeking and the demand for such service must originate with the laity. The laity cannot and should not expect to have such protective services supplied free, or at the expense of the tax levy, or through public agencies, except in case of proved dependency.

Lay organizations and public health services with medical guidance must and will urge such examinations as the best way for each person to participate in the benefits of preventive medicine.

Public health is a matter of private concern.

Preventive medicine can be made more effective through the family budget than by adding to tax-supported public functions.

Medical, educational and hospital organizations will have to teach physicians this new relationship, and set up and maintain standards of professional work which will produce results to command the respect and confidence of the public.

It appears to me that this offers the greatest opportunity yet presented for each party concerned, both physician and patient, to benefit from modern preventive medicine.

Health officers will admit, I believe, that where good public health services have been in effect, very little further reduction in death-rates can be accomplished until the individual takes on the burden of his own self-protection through the guidance and information received as the result of the health examination by his own family physician.

Health is an attainable objective for each member of the com-

munity only when individual services supplement public services. What we can severally do *for ourselves* at our own expense, when intelligently directed, will prove of even greater benefit and give us an even ampler freedom from disease and disability than has been granted us through those measures which have been carried out *for us* or done *to us*, without our personal conscious participation, at public expense.

Public health is now a matter not only for the health officer, but for each member of every community, and will in the future depend chiefly upon the breadth of personal interest and participation by the family and their medical adviser.

DISCUSSION

Dr. Howard C. Frontz (President of the Medical Society of the State of Pennsylvania, Huntingdon, Pa.): This very interesting and complete address of Dr. Emerson's has left very little room for discussion. However, I would like to give a few thoughts relative to his views on preventive medicine as we see it in the country districts of Pennsylvania. His argument and all that he says is ideal, and where there are large numbers of doctors who can develop that side of medicine it should be quite practical; but in the country districts, which now have fewer physicians for the population, quite a number of the men scarcely have any time to devote to it, and some still remember what they were taught in the medical schools, and that was to treat disease. I feel that the whole subject with us up in the country will be, first, a matter of education. We must have the doctor see that he has a responsibility to help prevent disease in his patients, and by his directing their lives and throwing out hints of how they can prevent illness he can be a great help to them. We also must educate the people along lines of health. We have in the last few years started in this state a large number of state clinics, which have been most valuable helps in tuberculosis work, along the very lines Dr. Emerson has alluded to, and this also has followed on with the well-baby clinics, and I may say in a county of 40,000 people we have two well-established well-baby clinics and one prenatal, and we are developing a service in prenatal work so that those worthy poor women who are in maternity and going alone without any help and with very little encouragement get help. We have them come to the prenatal clinic, and they are kept

under observation until time to be delivered, when they are admitted to the wards of our local hospital for delivery. These women are well cared for and under the control of a very competent obstetrician, and are receiving very valuable help and assistance in an important field of medicine. They are also directed how they shall live and care for their baby after leaving the hospital. Now, the matter of educating the doctors in some of the communities is difficult. There are hosts of doctors who are not interested at all in public health or preventive medicine. Personally, I think it is one of the most attractive branches of medicine. I am very much in sympathy with the idea of an annual examination and believe much good will be accomplished in time by it. In the country districts it is going to take time. The only examinations that gave us any idea of apparently well persons up to within a short time have been insurance examinations. Last evening I examined a man for life insurance who had never been ill or had a physician, and was apparently well until I examined his urine and found a fair amount of sugar in it, much to his surprise and mine, and of course that would reject him for insurance. It was a help to this man to know this in order to begin the modern treatment of diabetes early. The work of preventive medicine in some other fields has advanced in the country districts with a campaign against diphtheria by the use of toxin-antitoxin. This has been going on in Pennsylvania through the generosity of the Pennsylvania Department of Health, and in the county in which I reside we were able to immunize more than 2000 children of a population of 40,000 people. We were able to do this through a campaign of education. We asked people to come to meeting places, where we told them the story, before we attempted to give a single dose of the toxin-antitoxin. Much to our surprise people came in large numbers, and before we were through with the work many more desired to take it, but we were unable to give more at that time. That work could not have been put over except by the educational plan. I had an interesting experience. Recently a child visited a family in the county from a neighboring county; there were four children at home in the family, and this child became infected with diphtheria and died within two days.

The family did not seem to know where the child became infected; the four children had all received toxin-antitoxin five months before and were entirely free from that infection, and thus were saved. These are some of the things we are doing, and it has made a wonderful impression in this rural part of Pennsylvania. We therefore believe an educational campaign for preventive medicine must be kept up, first with the physician and then the people.

Dr. W. G. Turnbull (Deputy Commissioner of Health of Pennsylvania, Harrisburg): I do not know of anything that could be better, from the standpoint of Pennsylvania, than to arrange that the doctors could come here and hear papers such as Dr. Emerson has given us tonight. There is no question that the education of the physician is not only one of the very important things, but probably the most difficult single thing in getting ideal conditions so far as preventive medicine is concerned. This sounds rather bad to the laity, but to those of us who understand our conditions it is not as bad as it sounds. Unfortunately, the physician has to earn his living, he has been taught to treat disease, and people have been taught to go to the doctor when sick and not when they are well. Anything that smacks too much of state medicine is resented. Whether the education has to begin with the physician or people I am not sure. Personally, in the work I have seen done by the Department of Health, I believe we have accomplished more where the education has begun with the laity. The physician soon swings in if he finds the people are willing to pay for something he can give them. If we are asking him to do something that looks like cutting his income and forcing people to take something they do not want, he is not going to be very enthusiastic. In the work we started about a year ago on toxin-antitoxin immunization of children we found the physicians' passive resistance was the hardest problem we had to handle in some communities. It was only when we were able to show the physician that the work of the State Department of Health was really advertising for him that his work was increased that he became the most enthusiastic advocate of the prevention of diphtheria. So I am not at all sure that we will not have to do our first work and hardest work with the public, and we can rest assured then that the doctor will come along. I do feel if we could have all the physicians of Pennsylvania here and have such papers as we have heard tonight it would make the coming along very much easier. In the police era our state work was well organized. Under the administration of Dr. Martin and Dr. McLean, this was very much broadened, and we started out boldly in the educational era. That is going along. We are not sure whether it is done as thoroughly as in the police era, but we are making progress. We are trying to make the local community, as well as the individual, see that health is a local problem rather than a state problem. We are trying to organize well-baby clinics, prenatal and tuberculosis clinics and all the rest in such a way that they will ultimately be taken over by communities able to handle them and not become a permanent work of the state. Just how much of the work we can get done in the local communities is still a problem. At the present time the biggest work is the diphtheria immunization. In school work we have immunized 50,000 children. We

have already immunized more than that number of preschool age this fall. That work has just begun. From the fact that we have offered the physicians the opportunity of purchasing toxin-antitoxin through our department, we do know that a tremendous number of children are being immunized in addition to those that we are immunizing ourselves. We feel that this work is getting across, and getting across extremely well as an educational matter and not as a compulsion. It is gratifying to see how little resistance there is to it when once started. I was very glad to hear Dr. Emerson state that tuberculosis could be eliminated as a cause of death. Twenty years ago, leaders in the antituberculosis work believed that. Recently, we have looked rather pityingly on the deluded enthusiasts who believed in time tuberculosis would be wiped out. The statistics in the last year—I refer particularly to the statistics of the combined life insurance companies that have recently been made public—have swung us back, we have come to believe that possibly the enthusiasts of twenty years ago were right and that we are going to have tuberculosis wiped out as a cause of death. For the first time statistics seem to bear out the belief that ultimately it may be done. I want to express my extreme pleasure to Dr. Emerson for the paper which he has read.

Dr. E. Q. Thornton: We are extremely gratified to have a man of such wide experience and good judgment come before this society with this important communication. We have been instructed and delightfully entertained, and have had our interest in preventive medicine reinvigorated. That since 1885—based largely upon the Massachusetts records—the average span of life has been increased from forty years to fifty-five years, and that there is hope of still further extending this expectancy of life to sixty-eight years is most gratifying. That this has been largely brought about by improved method of sanitation and other preventive measures there can be no doubt, we should, however, give full credit to the wonderful advances in therapy, medicine, surgery, chemistry, bacteriology and to the many modern methods and instruments of precision now used for purposes of diagnosis and treatment.

Medical colleges of today are instructing their classes in matters pertaining to public health, and their graduates are practising the methods taught. National, state and county medical societies are educating the public how to conserve health. Another important factor in this educational work is the life insurance companies.

The people themselves have come to be interested in health and in measures to conserve it; they are no longer satisfied to be treated for their ills, but they ask for information as to how to keep well. They are no longer satisfied with verbal instructions, but they ask for specific

written directions as to food, clothing, climate, rest and other hygienic and sanitary measures. It is to the everlasting honor of such enthusiasts as Dr. Emerson, Dr. Abbott and other physicians who have devoted their lives to the study of preventive medicine and have educated and stirred to action physicians and the laity. It is our sincerest hope and belief that their earnest efforts will enable us to reach that goal of perfection for which the medical profession has ever striven, to lessen sickness and suffering and prolong the useful and pleasant days of man upon the earth which the Lord God hath given him.

Dr. A. C. Abbott: To all appearances the practising physician shall soon have to declare himself—to decide whether he be in belief a Malthusian, willing to let Nature take its course in matters of health and disease, or of the modern, progressive group who in the course of curing the sick feel themselves obligated to advise the well as to the means of preventing sickness.

Needless to say, questions of prevention are of prime importance to the lay public, and, fortunately, the lay public is now so well informed upon the progress made through the employment of preventive measures that it is coming to the point of demanding that physicians give it the full benefit of such measures.

I am not interested in fees or the financial side of the practice of medicine, but since that phase of practice has been injected into the discussion, it may be said that possibly in the future the practitioner may be paid not only for helping the sick to get well but for such advice as will prevent the well from getting sick. Whether the practitioner likes it or not, whether it pays or not, are aside from the question. The main point is that an informed public is demanding that such protective and preventive measures as one knows to be useful shall be employed for its benefit.

We are fortunate this evening in having brought before us so directly the possibilities of prevention in that large group of non-transmissible diseases hitherto regarded as beyond the influence of methods of prevention.

I trust Dr. Emerson may continue his activities in this important field.

Dr. Emerson (closing): I have in the past three years studied the extent to which preventive services are provided for the expectant mother. About 7 per cent of expectant mothers receive the benefit of adequate and nearly perfect facilities provided at public expense. This reaches only the lower group that cannot get such service unless provided for them by the public. In regard to the baby stations, tuberculosis and prenatal clinics there is only one place where they belong,

that is in the office of the medical practitioners of the community. There is only one place where they ought to be developed. The doctor's private office is the community's health center. Until the doctor makes his work there include health examination as well as treatment of the sick, he will not be fully using his privileges. The diagnosis of diabetes is commonly made first in the course of a life insurance examination, because it is not routine for doctors to make even urine examinations at an office call for diagnosis. Of the people who apply for life insurance, 1 per cent have glycosuria and 1 per cent are found to have some cardiac disease. With regard to the extension of the expectancy of life, our experience is based partly on Massachusetts data and partly on figures from the country as a whole. New Zealand has reached a life expectancy of 64.7, Kansas 60, although the country as a whole is credited with fifty-five years. Remember that Pittsburgh is the city with the shortest life expectancy in the United States and Washington with the longest. We are sometimes inclined to suspect that the determining factor is the relative industry of these communities. No one would accuse Washington of being called an industrial city. We are seeing in different parts of the country great demonstrations in the possibilities and technic of the prolonging of life; whereas in Kansas we have had a sturdy, independent race stock, no congestion of population, no slums, no large cities and—perhaps one may add as an item—a generation brought up, in the main, free from alcohol, with a continuous and consistent state health administration free from politics, we find the greatest longevity.

OPERATIVE TREATMENT OF SEPTIC THROMBOPHLEBITIS OF THE FEMORAL VEIN[1]

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Septic thrombophlebitis, irrespective of its site, always induces a serious clinical picture, frequently with unfavorable prognosis. Whether it be the lateral sinus, a tributary of the portal vein, the pelvic vein or veins of the extremities, insidious and progressive advance of the thrombus is the usual course. The clot laden with bacteria softens and small particles with engulfed microorganisms enter the blood stream. For a time the showers of microörganisms are apparently destroyed by the bactericidal agents of the body. Blood cultures are then not invariably positive. The condition is an intermittent bacteriemia. (Martin.) The prognosis depends upon many factors, but it is largely a question of the ratio between the number, type and virulence of the microörganisms and the resistance of the host. In general, however, the infection overwhelms the resistance: Organisms are arrested by the liver or lung, with resulting abscess formation therein, or they pass through these organs and give rise to metastatic abscesses in more remote parts. The pathetic and protracted picture of pyemia is the concomitant of this pathological process. No conservative therapeutic measures are successful. But since the serious results are due to the direct communication of the infected vessel with the blood stream the mind instinctively turns

[1] Read November 7, 1923.

to the possibility of early isolation of the infection by mechanical measures. This idea of surgical intervention in such cases has been practised to a limited extent. Although of relatively recent development it has some historical background.

It is said that John Hunter, in the eighteenth century, recommended compression of the vein above the thrombus in order to produce adhesive occlusion of the vessel above the obstruction. Apparently the first operation was performed by H. Lee, who, in 1865, cured several cases of purulent thrombophlebitis of superficial veins of the extremities by ligation of the veins above the affected areas. In 1877 Kraussold cured a case of pyemia following infection of an amputation wound by ligation of the femoral vein above the thrombus and resection of a portion of the venous trunk. Demons (1881) in a case of suppurative thrombophlebitis of the median basilic and cephalic veins successfully evacuated the thrombi. A few other early cases were reported in the domain of general surgery, but it was in the field of otology that ligation of the venous trunk and evacuation of the thrombus were shown to have real merit. Forty-three years ago Zaufal proposed exposure of the sinus, with evacuation of the infected thrombus and ligation of the jugular vein, in sinus thrombosis following middle-ear suppuration. He first performed the operation in 1884.

In gynecology the operation of "phlebectomy" has had a varied and not very encouraging career since it was first employed by W. A. Freund in 1898. He reported 2 cases of puerperal pyemia in which he excised the obstructed ovarian vein and thrombotic broad ligament. These operations, however, proved unsuccessful, as did 3 others reported by Bumm in 1902. For the frequent cases of puerperal pyemia, in which the veins are obstructed on both sides, bilateral ligation was proposed by Trendelenburg, who has reported several successful operations, 1 in 1911 with recovery after transperitoneal ligature of the vena cava and the ovarian veins. More recently Martens (1921) has operated upon 11 gynecological cases of thrombophlebitic pyemia, including 2 successful ligations of the vena cava. In the other cases the ovarian veins were ligated on one or both sides, and in addition

the hypogastric or the common iliac veins. Of the 11 patients 7 recovered. Martens advises an extraperitoneal approach.

With respect to the *intestinal tract* the literature contains a few cases in which thrombophlebitic obstructions of the mesenteric veins have been successfully operated upon. The first of these was reported by Brunner in 1907. Wilms, in 1909, in a case of pyemia originating from the appendix, ligated the mesenteric veins at the angle between the cecum and ileum, with the result that the chills were promptly arrested and the patient made a good recovery. Braun, in 1913, elaborated this operation by ligating the ileocolic vein shortly before its entrance into the superior mesenteric and resecting the thrombosed vessel with success in 2 cases. Weil, in 1920, reported a case of pyemia of appendiceal origin cured by a similar procedure.

The femoral vein is a frequent site of thrombosis and occasionally of suppurative thrombophlebitis, yet surgical intervention is infrequent. In fact, operative efforts in such cases have been so rarely reported that one wonders whether the possibilities of benefit by operation have not been generally overlooked. I am led to believe that many cases susceptible of benefit by operative treatment have been conservatively observed until their deaths, because of the conviction that signs of sepsis always indicate a general infection which cannot be controlled by local measures. Gluck, in 1896, reported several successful cases, in one of which there was thrombophlebitis of the long saphenous vein seven weeks after abortion. The patient suffered from high fever, chills, icterus, enlargement of the liver and spleen and weakened heart action. The treatment consisted in ligation of the common femoral vein, and the patient recovered. A case of saphenous thrombosis was successfully operated upon by Müller in 1909, through ligature of the femoral vein. Riedel, in 1911, reported a case on which he had operated many years before removing a septic thrombus from the femoral vein.

In this country, W. Greiss, in 1913, observed septic thrombosis of the femoral vein four weeks after an operation for gunshot wound of the intestine. He exposed and incised the thrombosed

vein, removed the thrombus and resected six inches of the vessel. The patient recovered. Rost, 1916, in 2 cases where amputation of the leg was followed by septic manifestations suggestive of thrombophlebitis, ligated the trunk of the femoral vein with recovery.

Rosenstein, as the result of considerable experience, recommends high ligation of the external iliac vein in certain cases of thrombosis of the femoral vein. His first successful case was a man, aged fifty-four years, with varicose ulcer of the leg and thrombophlebitis of the right femoral vein. The femoral vein was ligated, with extirpation of the entire thrombotic trunk. In the second patient, a woman with mastitis and thrombosis of the femoral vein, ligation of the common iliac vein was performed, followed by extirpation of the entire thrombotic portion as far as the saphenous vein. The outcome was good. The third case was one of progressive thrombosis in a woman, aged twenty-eight years. The ligature was applied in uninvolved tissues directly below the bifurcation of the common iliac vein and the thrombotic venous segment was resected as far as the saphenous. The patient made a good recovery. Another case, in which the upper extremity was involved, occurred during Rosenstein's service in a field hospital and concerned a young soldier whose left arm had been amputated. Subsequently disarticulation at the shoulder-joint had to be performed on account of infection. After temporary improvement general sepsis developed. The subclavian vein was exposed below the clavicle and was found to be thrombosed at this site, with inflammatory changes in the surrounding tissues. Accordingly a ligation was done close to the origin of the vessel and the patient made a prompt recovery.

A similar case was observed by one of us (E. H. P.):

On April 11, 1918, a French soldier was admitted to Evacuation Hospital No. 1, A. E. F., presenting multiple wounds of leg, forearm and thorax. The forearm presented advanced gas-bacillus infection. The patient was in profound shock.

After reaction from shock, disarticulation at the elbow was

done under local anesthesia without moving the patient from bed. Intravenous administration of 600 cc of 5 per cent sodium bicarbonate solution, Bull's serum, 10 cc, was injected through the infusion tube. Much improvement. Hemothorax.

April 13. Temperature septic since admission, reaching 103°. Pulse, 100 to 110. Bull's serum, 10 cc. Red blood cells, 1,600,000. Hemoglobin, 38 per cent. Transfusion into internal saphenous vein. Local evidence of brachial vein thrombosis developed. Without moving the patient from bed the vein was ligated in the axilla, incised downward and the suppurating clot removed. The wound was left open and treated by the Carrel-Dakin method. Progressive improvement. Evacuated May 15.

In 2 cases of septic thrombophlebitis of the great saphenous and femoral veins reported by Santrucek, in 1920, the septic manifestations subsided at once after ligature of the femoral vein. In a third case, where there were as yet no symptoms of propagation of the septic material through the blood current, the femoral vein was ligated as a prophylactic measure. The patient made a good recovery. Such prophylactic venous ligation and resection is advocated by Hosemann on the basis of 7 successful cases.

Postoperative femoral thrombosis following aseptic celiotomy is quite frequently noted and much interest is attached to this condition on account of the obscurity of its origin and the occasional serious sequelæ. Clark found 36 cases of thrombosis in a series of 3000 celiotomies. The theories as to the origin of such thrombi have been collected and elaborated by Albanus, Aschoff, Conner, Clark and others. They need not be reviewed and only a brief reference will be made to this phase of the subject. To quote Aschoff: "Thrombosis is the function of a number of variables. There is not a single cause, but quite a number of different conditions which are closely related to the occurrence of thrombosis. Among these may be mentioned here, first, changes in the blood plasma (diminished or increased coagulability); second, changes in the blood elements (increased or

diminished powers of agglutination); third, changes in the blood flow (slowing and formation of eddies), and last, changes in the vessel wall itself (endothelial damage). An inquiry into the mechanism of thrombosis shows that sometimes one factor, sometimes another, plays the principal rôle." Clark concludes that postoperative thrombophlebitis is referable to traumatism exerted upon the deep epigastric vessels during the operation. Originating in the deep epigastric vein, the primary thrombosis, he believes, is slowly propagated along the vessel until it reaches the external iliac, where it gives rise to a retrogressive thrombus in the femoral vein, usually on the left side. While this explanation is doubtless applicable to a certain proportion of cases, it does not readily account for thrombosis following a high incision or for thrombosis of the left femoral following operations through the right abdominal wall. Some years ago we had a case which is of interest in connection with Clark's theory. Fatal pulmonary thrombosis followed an operation for right inguinal hernia, during the course of which the deep epigastric vessels had been ligated and divided. Autopsy showed a thrombus in the pulmonary artery which completely obstructed the vessel. The upper part of the femoral vein contained fluid blood, the walls were smooth and glistening except a large valve immediately beneath Poupart's ligament. Three inches below Poupart's ligament the vein was found to be occupied by a thrombus which extended downward for two or three inches. The upper part of the thrombus was soft and very friable; the lower portions were more cohesive. It was assumed that the thrombus originated in the deep epigastric stump and extended thence into the femoral, the upper portion of the femoral thrombus giving rise to the pulmonary embolus. (New York Hospital History, No. 209768, January 4, 1917.)

The rare cases where the postoperative thrombus is loaded with bacteria, constituting a septic thrombus, as in the case to be reported, must be explained as due to hematogenous infection with localization and colonization of pyogenic organisms in the vessel wall. (Matas.)

The clinical picture of septic thrombophlebitis has been described

by Matas and will not be discussed. When the veins of the extremities are affected the diagnosis should not, as a rule, occasion much difficulty because of the intensity of the local and systemic signs and symptoms.

The case which is the basis of this paper follows:

Male, aged thirty-four years; English; married; janitor. Admitted February 16, 1923, to Second Medical Service, New York Hospital.

History. For years has had attacks of indigestion. Attacks of pain came on two or three hours after eating. Had occasional night attacks. These were relieved by belching or vomiting. No severe attacks for past six years but a feeling of epigastric fulness and discomfort after meals. Had tarry stools two weeks before admission.

P. X. Poorly nourished, intelligent man. Heart, lungs and abdomen negative.

Urine. Negative.

Blood Count. Normal.

Clinical Diagnosis. Duodenal ulcer. This was corroborated by *x*-ray findings.

Operation. Excision of ulcer of duodenum. Appendectomy in course.

Pathological Report. Small ulcer of the duodenum with round-cell infiltration.

Course. For four days after operation the temperature was slightly elevated; it then came to normal and remained normal until the sixteenth day after operation. Wound healed by primary union. On the sixteenth day the temperature rose suddenly to 104° and then ran a septic course from 99° to 105° until April 23, the sixty-third postoperative day. During this time the patient had gone down hill steadily, was having chills and sweats, but complained only of headache. Repeated blood counts varied between 10,000 and 17,000 with a polymorphonucleosis. He was markedly septic and very weak. He was repeatedly examined without anything definite being found until March 28, when evidence of

phlebitis of the internal saphenous vein was noted. Blood culture taken on March 26 showed hemolytic streptococci. March 29 and April 3 blood cultures sterile. One on April 12 again showed hemolytic streptococci.

On April 23 (sixty-third postoperative day), under local anesthesia, the left femoral vein was exposed, ligated, opened and the thrombus removed.

Operation. Three-inch incision over the upper portion of the femoral vein; vein exposed and opened. Pressure was maintained digitally over the region of the inferior vena cava about one inch above and to the right of the umbilicus. There was a friable thrombus which broke into fragments during removal. One segment projected like a cork into the profunda vein. When this was removed there was free bleeding from the profunda. An aspirator and also a gall-bladder clamp were introduced proximally through the opening in the femoral vein, but no clot obtained from above. The total length of the thrombus removed was about three inches. Internal saphenous ligated and the femoral ligated at each end of the wound. The profunda was also ligated. Wound left open. Pathologist reported that thrombus contained Gram-positive cocci arranged in pairs and chains.

Following operation the patient improved wonderfully; had no more pain, chills or sweats. Temperature dropped to a lower level, 99° to 101°, and blood cultures were repeatedly negative. On May 14, under local anesthesia, a left popliteal abscess containing hemolytic streptococci was incised and drained. Following this the temperature remained normal and improvement continued.

The patient was kept in bed six weeks in order to favor the development of venous channels to minimize the effects of the femoral obstruction. At first the extremity was markedly swollen; this gradually diminished, and since getting up and about there has been little swelling and no subjective symptoms. The patient was examined on October 1, five months after operation. His general health is excellent and he has gained twenty pounds. Returned to work one week after discharge. He has no pain in

the limb. There is slight swelling at the end of the day. Wears a bandage. Measurements: Right thigh, 18½ inches; left thigh, 20 inches; right calf, 12½ inches; left calf, 13¼ inches. Slight pitting over dorsum of foot; no local tenderness or cyanosis.

Some features of the case are worthy of review:

Patient septic, losing ground rapidly with positive blood culture and essentially negative physical examination except a left femoral phlebitis.

The phlebitis was presumably secondary to some undiscovered focus. At the time of ligation we hoped that it was the only active focus. That this was the case is supported by the subsequent clinical course.

The low-grade fever which the patient ran following the second operation received full explanation when a popliteal abscess was evacuated. Temperature then immediately dropped to normal and remained normal. This popliteal collection was evidently the result of a slow, suppurative, retrograde thrombophlebitis from the site of ligation downward.

Although the operation was successful the operative procedure could have been improved in several details, as will be noted later.

Conclusions. It is often possible to explain the occurrence of suppurative thrombophlebitis. For instance, a large vein in proximity to a suppurating area so that pyogenic bacteria penetrate its wall, as the lateral sinus in mastoiditis; or involvement of small efferent veins in a pus focus, with extension of the thrombi into the larger venous trunks, as pylephlebitis complicating acute appendicitis. Yet in many cases the explanation is impossible.

A septic thrombus frequently is at first a local and solitary focus of infection. From it microörganisms are passed into the general circulation and may be recognized by blood cultures. The clinical picture is one of septicemia. This condition prevails for a variable period before secondary abscesses or septic infarcts develop. Under such conditions isolation of the infection by proximal ligation of the vein may arrest the disease. The prog-

nosis under such treatment is most favorable in early cases, especially when blood cultures are not constantly positive, indicating an intermittent bacteriemia. When the thrombus is one of multiple foci in a general infection the infection is beyond control.

A patient who presents the signs of phlebitis in the presence of unexplained, persistent, septic temperature and positive blood cultures is entitled to operation upon the involved vein if this is accessible. One never hesitates to drain an abscess, and the same general rule should apply to infections in veins, with the added indication that an infection in a vein is infinitely more dangerous, because it is so situated as to threaten the general blood stream.

The operation can be done on veins of the extremities under local anesthesia without serious discomfort to the patient.

As to the question of delaying ligation in the presence of suppurative thrombophlebitis because of doubt as to the competency of the collateral circulation, let it be borne in mind that a thrombosed vein is no better than a ligated vein in the matter of carrying on the circulation.

In the surgical treatment of septic thrombophlebitis the necessity for central ligature of the healthy venous trunk is obvious, and the guiding rule must always be to operate in uninvolved tissues. Hence, in thrombosis of the femoral vein the external iliac vein should be exposed. It should be determined by inspection and careful palpation, and if necessary by incision, that the exposure is above the thrombus. Until the infected vein has been occluded by ligature it is wise to interpose some block above the vein on the cardiac side to minimize the chances of setting fragments free. Therefore, digital pressure should be maintained on the vena cava through the abdominal wall during these preliminary procedures. Having demonstrated that the exposure is above the thrombus the vein should be ligated. It is better, as pointed out by Rosenstein, to ligate a few centimeters too high than to work in diseased tissues. In some cases this is all that can be done either by reason of the condition of the patient or the situation of the vein. As has been shown, a number of cases have been cured by this procedure. But, in general, the

thrombus should be removed. This may be done in several ways: The vein may be split downward and the upper end of the thrombus identified and the incision continued if possible the full length of the thrombus, which should be removed with or without the vein wall; or the unopened vein and thrombus may be removed together. Timely interference is always advisable as soon as the condition of septic thrombophlebitis has been recognized. In view of the fact that the operation in itself is practically devoid of danger, that all other measures are inefficient and that the disease in severe cases is almost invariably fatal, surgical interference is definitely indicated.

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DISCUSSION

Dr. Eldridge L. Eliason: This paper of Dr. Pool's has been very admirably presented, has covered the historical features and has brought the matter up to date. There are two or three features to be looked at from the standpoint of the surgeon which are rather interesting. Dr. Pool brought out the fact that in some of these cases we cannot determine the source of the infection. Other cases of phlebitis come from continuity. When the thrombosis occurs in the jugular vein it is by extension, the infection coming from the mastoid. Some infections in the femoral vein come from infection in the immediate neighborhood of that vein. But, on the other hand, the clinical picture we see of phlebitis following operations of various types is inexplainable. We have a perfectly clean case and we get phlebitis of a low-grade inflammatory type, usually in the left limb—why, we do not know. It is conceivably due to lowered resistance, lowered blood-clotting time, a low-grade infection in the blood stream or to an anatomical formation of the vein. Dr. Pool brought out the question of an intermittent bacteriemia. I have been much interested in that phase of the clinical picture.

I have only seen 4 or 5 cases of thrombophlebitis that came under my care from operation: 2 were jugular; 1 I opened and removed a clot that

extended from practically the jugular foramen to the clavicle. This child had a negative blood stream. The only case of femoral phlebitis that came under my care was secondary to an infection in the thigh, to which I was called in an emergency. The infected clot had ulcerated through and the man was bleeding from a ruptured vein. That man had a negative blood stream and the patient died of mediastinal abscess. The fact that these cases have such findings as regards the blood answers the question as Dr. Pool answered it, namely, that the medical antiseptics are of no avail. In the first place it is not necessarily a blood-stream infection. The infection, as we see it at the time of thrombosis, is a localized plugging of that vessel and the blood stream will not carry the antiseptic fluid.

In regard to the treatment of these cases, where it is possible excision should be done, with the precaution Dr. Pool brings out of going into normal tissue and ligating the normal vein. If a personal experience be pardoned, a case I saw of this type, the vein was so diseased that it was crushed off and a hemostat put on it. It had to be ligated above. Had I known as much then as now after hearing Dr. Pool's paper I would have gone higher at first. Some of these cases do get well, however, in the absence of surgeons. Thrombophlebitis occasionally does recover. I saw a case recently in which the postoperative complication resulted from appendicitis. A severe, far-advanced condition was found. Nothing was done. That was six months ago, and the man is well today. I think we have all been very fortunate in having Dr. Pool to present this subject to us, and I am sure I have been greatly benefited by it.

Dr. Richard C. Norris: What has been said by the general surgeons as to frequency, treatment and results is applicable to gynecological practice. In obstetrics we meet especially with pelvic and femoral thrombosis. As I look back through my experience I have never seen occasion for an operation in the puerperal state. Such cases almost always get well. It is rare for them not to. At the time Trendelenburg wrote his paper we were all practically interested in this, and there were few men in Philadelphia willing to attempt the formidable operation of ligation of veins in the pelvis at a time when the clinical course of the disease did not justify an abdominal operation. It takes a very bold surgeon to tackle an operation of that kind sufficiently early to do any good, and it is commonly followed by a very high mortality. The trouble in these cases is the question of diagnosis and degree to which the thrombosis process has extended. He stands on very uncertain ground—too uncertain to warrant exploratory laparotomy to determine what procedures should be carried out after entering the abdominal cavity. There have been periodic waves of surgical enthusiasm to attack intra-abdominal phlebitis, but with little

success. The time may come when our diagnostic methods may reach such perfection that it will enable the surgeon to stand on more certain ground when he attacks these cases from the surgical standpoint. The laboratory research man must be the forerunner. He must help us to earlier and more exact diagnosis. Whenever the subject pops up for discussion it has always seemed to me that if we could get the research laboratory to make further progress working upon the lower animals, the surgeon might get some real help. To experiment and do research work on human beings afflicted with this disease is a problem surgeons should not attempt. In superficial cases, where suppurative or necrotic changes are unmistakable, we should do operative work. The crux of the situation in abdominal cases is early and precise diagnosis. As an etiological factor in some cases of femoral phlebitis, traumatism of the abdominal wound by retractors has been mentioned. This cannot always be avoided.

Dr. Charles P. Noble: In line with Dr. Norris' remarks, and speaking, of course, from memory, I would like to refer to my experience as a gynecologist with phlebitis: First, as to the obstetrical side. My experience in obstetrics has been much less than Dr. Norris's, because it stopped years ago; but still, as a gynecologist, one was called in consultation in complicated obstetrical cases. As to Trendelenburg's procedure, I confess that my conservative tendency was greater than that of the gentlemen who tried it out, and I have no experience to report along the Trendelenburg line. My judgment as a surgeon and obstetrician was that in these cases of puerperal infection with pelvic phlebitis the patients were too sick to withstand both the disease and the surgeon, should he attempt the formidable procedure proposed. I felt that patients had a better chance of recovery without operative interference in the abdominal cases than with it. In gynecological practice, curiously, in a very long experience, particularly in involvement of the femoral vein, I have never seen a phlebitis that suppurated, and I do not recall a single one that died either with or without embolism. Of course, I have had patients die of embolism after operation, but in those patients no phlebitis had been recognized.

Dr. Edward J. Klopp: Apparently there are two forms of phlebitis, the toxic and the septic variety. The ones due to nonpyogenic organisms do not enter surgery from the operative standpoint, as do the cases due to pyogenic. I should not say, however, that those due to nonpyogenic organisms do not interest us at all, because they do, so far as diagnosis and treatment are concerned. The diagnosis is not always clear. The diagnosis of femoral phlebitis is not always made early. One frequently finds that the pulse-rate is increased some days before rise in temperature

and before there is pain in the groin. One also frequently finds that there are enlarged lymph glands even though there is no swelling and pain in the thigh or foot, where it very often begins; in the absence of swelling and pain in the thigh and calf of the leg, and so on, we sometimes fail to make an early diagnosis of phlebitis of the femoral vein. A word in regard to the treatment of nonpyogenic thrombophlebitis of the femoral vein: we use ice-bag, bandage or splint. Perhaps the bandaging of the leg is the worst thing to do; if there is a great deal of pain, rest the thigh on a pillow; there is not much danger of dislocating clot. There is a great deal of danger of dislocating the clot if you bandage the thigh. The cases due to pyogenic organisms interest us considerably. We have seen a small number of cases due to pyogenic infection involving the femoral. We have seen many involve small veins, especially the facial vein in connection with furuncle or carbuncle of the upper lip. The proper treatment is high ligation of the vein, to prevent the infection from extending to the base of the brain.

A few words in reference to the diagnosis of thrombosis of the cavernous sinus. This is something that many of us overlook; even oculists sometimes mistake the condition for brain abscess. The rhinologists and otologists sometimes mistake it for frontal sinus infection. It behooves us to recognize it when we have a case of thrombosis of the cavernous sinus. I was much interested to hear Dr. Eliason say a case of ruptured phlebitis died from hemorrhage. I understood those that ruptured spontaneously seldom bled profusely, although if opened by the surgeon they might discharge pus and dark blood. Of course, there are two types of suppurative thrombosis: the one where there is an ulceration through the wall involving the tissues surrounding the vein (these cases necessitate an incision through the skin only, and do not require an expert surgeon like Dr. Pool); the other, where collections of pus appear at intervals separated by clots; these are the cases that usually require resection or evacuation of a certain number of clots and drainage.

Dr. Pool (closing): I seem to have put emphasis on the wrong features in my paper, because, judging from the discussion, it has been assumed that I advocate the Trendelenburg operation for pelvic thrombophlebitis and ligation of the ileocolic vein for certain complications of appendicitis. Such is not the case. I simply referred to these features in passing in review the various efforts that have been made toward operative treatment in various types of thrombophlebitis.

I was led to write this paper because the analysis of the literature and conversation with surgical friends led me to believe that the possibility of surgical intervention in septic thrombophlebitis of readily accessible veins

has been often lost sight of, with consequent loss of lives that might have been saved. It is too often not recognized that in many cases there is at first an infected plug in the vein from which organisms enter the circulation. Such cases may often be cured if this proposition is accepted and early operative treatment instituted. Recently, I have seen two such cases. One was a man whose arm had been amputated; he was profoundly septic and it never occurred to them to examine the brachial vein, which was large and solid. Autopsy showed a septic thrombus with infection extending along the vein into the mediastinum. It is probable that if this had been recognized early and the vein ligated high the patient would have recovered.

One other feature has to deal with the technic of the blood cultures. When a series of blood cultures are negative, although the clinical picture is one of bacteriemia, a check should be made on the culture media.

THE REACTION OF THE PARATONSILLAR TISSUES TO TONSILLECTOMY: A STUDY IN THE ETIOLOGY OF POSTTONSILLECTOMY PULMONARY ABSCESS[1]

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AND

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Introduction. Since the appearance, eleven years ago, of Richardson's[1] paper there has developed, especially in the last few years, a deep interest in the incidence of lung abscess as a sequel to tonsillectomy. A recent comprehensive study of the situation, based on a widely circulated questionnaire, has been made by Moore,[2] who gathered reports of 202 cases. Among his conclusions are the following: "The vast majority of cases are of inspiratory origin because of (*a*) time of development and (*b*) involvement of the lower lobes of the lung in 60 per cent of the cases (right lower, 41 per cent; left lower, 19 per cent), being almost the same relative incidence as in cases of inspired foreign bodies.

"Blood-stream transmission of infected material, causing pulmonary abscess, occurs, but in a relatively small number of cases. Lymphatic extension is a rare mode of infection."

Possible Pathways of Infection. With this dire sequel occurring so frequently, and it cannot be questioned that it happens oftener than even Moore's paper would lead us to think, the thoughts of all interested must turn toward the reason or reasons for its occurrence and the adoption of methods of prevention.

[1] Read December 5, 1923.

The infection must reach the lung either (*a*) by way of the trachea and bronchi (bronchogenic), (*b*) by way of the venous system (hematogenic) or (*c*) by way of the lymph channel (lymphogenic). The writers of this paper instituted this study with no preconceived theory as to which pathway is the preponderating one, but they were of the opinion that the likelihood of the inhalation method of infection has been taken too much for granted. The fact that in Moore's series of 202 cases 39 (approximately 20 per cent) were secondary to operation under local anesthesia strengthened this view.

Influence of Complete Tonsillectomy upon the Incidence of Lung Abscess. It was in 1912 that attention first was drawn to lung abscess as a sequel to tonsillectomy. And it was about that time that the so-called "tonsillotomy" was being discarded in favor of complete removal, including the capsule. Possibly this is not a pure coincidence. Dean[3] says, "In my experience the infections have been much more frequent and severe since I have been performing tonsillectomy and not tonsillotomy." The infections he refers to are those in the immediate region of or adjacent to the tonsil. The incomplete tonsil operation, whether performed by guillotine, punch or what not, invariably left behind a mass of tonsil tissue walled off from the throat by a very firm and capable capsule. The raw surface left after operation was composed only of tonsil tissue with its associated vessels. No injury had been inflicted or infection implanted upon the muscle or other deeper structures of the pharynx, and no vessels of the tonsillar and pharyngeal region were traumatized or severed. In those days the use of a general anesthetic was universal and no suction appliance was in use, and yet posttonsillectomy lung complications were neither noted nor reported.

Lung Infection of Bronchogenic Origin. Let us consider for a moment the question of air-route infection. A fair fundamental question would be this: Is the lung very susceptible to infection through the air passages? The answer is, It is quite probable that the lung possesses inherently a highly protective margin of safety. This is evidenced by the fact that many injuri-

ous objects, notably bacteria, are daily being inspired under many and varying conditions with no deleterious result. General anesthesia offers a good opportunity for plugs of mucus and clumps of bacteria to enter the respiratory spaces, thousands of cases with dirty mouths are being etherized, but postoperative pneumonia is not as common as the possibilities thus afforded would lead us to anticipate. For example, in Cutler and Hunt's[4] 1920 series of 1604 operations there were but 7 cases of bronchopneumonia and 1 of lobar pneumonia occurring as complications. In these 8 cases local anesthesia was used once (in the lobar pneumonia case), gas-oxygen four times and ether three times.

Moreover, the production of experimental pneumonia by the atomizing of bacteria into the trachea has proved very difficult of attainment. It has succeeded regularly only when very large doses or highly virulent organisms have been used and when they have been introduced by puncturing the trachea through the skin of the neck.

The question might be put more broadly: Are unbroken or even broken mucous membranes reasonably subject to septic invasion? For example, is an abscess of the nasal septum not a very rare condition? How many rhinologists have seen an abscess of a turbinated body, and this even considering the abrasions and open pathways for infection caused by the common habit of finger removal of crusts from the nose? How many cases of foreign body in the bronchial tree, the esophagus, the eye, the urethra or the intestines are followed by abscess? Very few! Of course, the bronchial and pulmonary mucosa may be less tough than that in the other locations mentioned; but even granting this, when a lung inhales some septic material in which are microörganisms to which the individual is probably partly immune, why should we necessarily assume that this inspired material is the cause of an abscess to the exclusion of other possible sources of infection?

Lung Infection of Hematogenic Origin. On the other hand, postoperative pulmonary embolism, following surgical procedures in the abdomen and elsewhere, is a common condition. Many of these cases are badly shocked and die promptly, because the

embolus comes from a large vessel and blocks off a large branch of the pulmonary artery and a large area of lung. Even if the embolus is a septic one, death in such cases usually occurs before an abscess has time to form.

In the field of general surgery the thoughts of investigators are turning decidedly toward the embolic nature of postoperative lung complications. A careful and exhaustive study recently has been made by Cutler and Hunt[5] of a year's cases in the surgical wards of the Peter Bent Brigham Hospital, where a specially careful watch has been kept for the appearance of postoperative lung lesions. As the result of improved methods of case-watching and study, the percentages of such complications found has more than doubled in five years. Along with this naturally has come a drop in mortality percentage, because many small lesions have been discovered, lesions which appeared and disappeared with but few symptoms.

During 1920, 1604 cases were operated upon, and "63 of these patients developed a pulmonary complication that might be attributable to the operative intervention or to the anesthetic. There were 5 deaths among these 63 cases. Thus in 3.93 per cent of the cases (1 in 26) a complication developed and 0.3 per cent (1 in 321) patients died from one of the complications."

In this group of 63 there occurred 1 case of lobar pneumonia, 7 of bronchopneumonia, 16 of bronchitis, 2 of exacerbation of tuberculosis, 2 of pleurisy, 1 of empyema, 32 *of pulmonary infarction* (italics ours) and 2 of pulmonary embolism. Thus, even in a general surgical clinic approximately 1 case in 50 was complicated by an infarction of the lung, which shows that this form of pathological lesion occurs more frequently than is generally thought.

Among their conclusions are the following:

"Irritation and aspiration due to inhalation anesthesia may be the cause of a small percentage of postoperative pulmonary complications.

"The fact that these complications occur with local anesthesia, with inhalation anesthesia in the most expert hands and in a definite relation to the mobility of the operative field, is taken

as evidence against the importance of the irritation of inhalation anesthesia in the production of these lesions."

W. J. Mayo[6] states that septic emboli from the operative field are a common cause of secondary pulmonary complications. He laments that these complications are too frequently attributed to the anesthetic, and says that they are quite as frequently found in cases in which local anesthesia is employed. Many other surgeons could be quoted to the same effect.

Among otolaryngologists the possibility that these lung lesions are of embolic origin is not entirely a new one. Of those who might be quoted in this respect are Richardson, Porter, and Simpson and Noah.

Richardson[7] in his 1912 report describes 2 cases under the title of "Septic Infarct of the Lung."

Porter[8] says: "There exists a complicated network of veins, the plexus tonsillaris and the plexus pharyngeus, which cover the outer walls of the pharynx and the tonsillar cavities, receiving blood from the tonsils, tonsillar pillars and pharyngeal walls. These vessels are exposed and always injured even in the gentlest operative work. This too is a field impossible to protect from germ-laden saliva and pus. It is therefore not difficult to believe that infected emboli could be dislodged from veins thrombotic from trauma and infection, located in an area active in every attempt at speech and swallowing. I feel that there can be no doubt that infected emboli travel direct to the lungs and cause certain cases of pulmonary abscess."

Simpson and Noah[9] state, "The writers believe that aspiration does not account for the production of lung abscess in the 2 cases reported." They conclude: "1. During or following operation septic material enters the veins, passes through the right heart to the lungs and there finds, in the presence of a tuberculous lesion, suitable soil for the production of an abscess.

"2. The possibility of aspiration of infected material as a cause of pulmonary abscess is not to be denied, yet we believe that more cases occur as a result of hematogenous infection than is generally supposed."

Answers to Arguments Favoring Bronchogenic Origin. A common argument in favor of the aspiration of infected matter as a cause of lung abscesses is that in location (preponderatingly on the right side and in the lower lobes) they correspond quite closely to that of foreign bodies. We believe this argument is not as definitive as appears on the surface, for the following reasons:

1. Foreign bodies have no monopoly of localization in the right lung and its lower lobe. Pulmonary emboli, especially massive ones, seek the right side to even a greater degree than do foreign bodies. An abstract[10] of an article by Capelle[11] states that: "Pulmonary infarction and embolism (*massive*—authors) was stated to have occurred in 15 among 10,000 operations, but many cases called postoperative pneumonia are really instances of small pulmonary infarctions. A distinguishing sign is the bright-colored blood expectorated as compared with the rusty sputa of pneumonia."

Then follows a sentence having marked bearing on the question we are discussing: "Thirteen presented changes in the back and front of the lower pulmonary lobes, and in only 2 were the signs restricted to the left lung." Thirteen out of 15 were in the right lung and all of them in the lower lobe! Does this not cast doubt upon the location of lung abscesses proving their inhalation etiology? So much for *large* emboli.

In searching the literature for data as to the localization of *small* infarcts we were unable to find definite figures. A letter of inquiry to Dr. Elliott C. Cutler[12] brought a generous and prompt response, and we are able to give his unpublished figures on this point. We herewith desire to express our deep appreciation of his kindness. His table is as follows:

DISTRIBUTION OF POSTOPERATIVE PULMONARY INFARCTION.

	1920 cases.	1921 cases.
Right upper lobe	0	2
Right middle lobe	2	1
Right lower lobe	14	7
Left upper lobe	0	0
Left lower lobe	5	4
Both lower lobes	2	1
Right upper and middle lobes	1	0
Left upper and lower lobes	1	0
Totals	25	15

Cutler had accurate data on 40 of his cases. Of these 21 were in the right lower lobe only and 9 were in the left lower lobe only (21 to 9); 30 of the cases involving more than one lobe were located in the right lung and 13 in the left lung (30 to 13).

Welch[13] states that: "The course followed by an embolus in its travels is determined by purely mechanical factors, of which the most important are the size, form and weight of the plug; the direction, volume and energy of the carrying blood stream; the size of branches and the angles at which they are given off, and the position of the body and its members. In accord with these principles we find emboli in the lower lobes of the left lungs oftener than in the upper, and in the right lung oftener than in the left, the right pulmonary artery being larger than the left."

The size of the right pulmonary artery and right lung is to that of the left pulmonary artery and left lung as 4 is to 3. Therefore, an embolus leaving the right heart, other things being equal, would have 4 chances of going to the right lung to 3 of going to the left, or in 100 cases 57 would go to the right and 43 to the left.

2. Another factor could be the course of the two pulmonary arteries as and after they leave the pulmonary aorta. Possibly the right pulmonary artery is in more direct line with the pulmonary aorta than is the left. We are at present studying this piece of anatomy, but are not yet ready to report.

3. The inhalation of bloody throat secretions during tonsillectomy is, in our belief, not precisely comparable to the dropping or inhaling of a foreign body into a bronchus. In the great majority of cases foreign bodies enter while their host is in an erect posture, and in dropping down the trachea they naturally would tend to enter the wider and more vertical right bronchus. On the other hand, during tonsillectomy, under ether, with the exception of certain parts of New England, the patient usually is in a recumbent position and gravity could have little or no bearing on the problem. Whatever enters the trachea is drawn in during inspiration and consists of frothy, bloody mucus and tonsil crypt contents. It moves up and down the larynx and trachea during respiration, and when a collection of this material

reaches the tracheal bifurcation it is almost as likely to be inspired into the left lung as into the right.

4. A final argument against the general acceptance of the respiratory theory of these lesions is the fact that so many are reported as being sequential to operation under local anesthesia.

With these facts in mind it is at least conceivable that the inhalation theory, based upon a wider right bronchus with a more direct course in relation to the trachea, should be relegated to a less important place than it hitherto has occupied in the minds of otolaryngologists.

Reason for and Plan of Present Communication. With these thoughts as a working basis, it occurred to us that a minute study of the pharyngeal and cervical region following tonsillectomy would tell us exactly what happens there as a result of the operation. We hoped that examination of the peritonsillar tissues, and especially of those venous and lymphatic tissues which drain the tonsil area, might afford information as to the probable location of the *fons et origo* of lung abscess. We felt that we would be studying a region into which septic tonsil contents are squeezed during operation, which would be traumatized during operation, into which needle-borne septic sutures frequently are carried during operation and which is wide open to constant microörganismal infection after operation. Such conditions are ideal for the formation of septic thrombi which later could become detached and pass down the internal jugular vein into the right heart and thence into the lung. We felt also that there is hardly any place in the body where thrombi would be more likely to become detached, because the contraction of the tongue and throat muscles during swallowing certainly would promote the loosening of vein clots and the starting of them on their way down the jugular, through the heart and into the lung.

The present communication is based upon the results obtained from operations upon animals. Our experiments were as follows: One animal we devoted solely to the identification of the tonsil lymph node, using Gerota's technic, as employed by Wood[14] in his study of the same structure in man. The others were ton-

sillectomized. In one of them we removed one tonsil and left the other undisturbed, so that the tissues of the two sides subsequently could be compared. In the remainder both tonsils were removed: On one side two sutures were introduced and tied, while on the other an attempt was made to infect the raw surface by swabbing

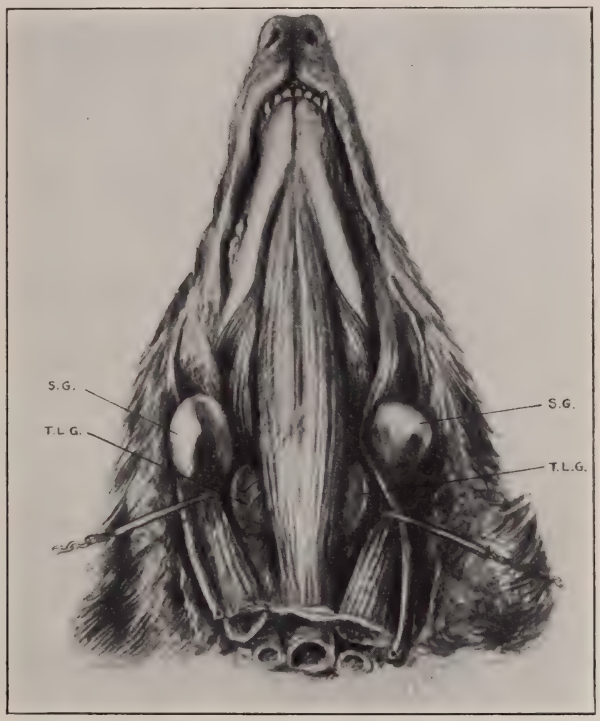


FIG. 1.—Author's drawing of a dissection of the neck, made after the tonsil region had been injected with a turpentine-ether solution of Berlin blue. The tonsil lymph node is shown, with its afferent lymph vessels. These vessels were traced back to the peritonsillar connective tissue. *TLG*, tonsil lymph node; *SG*, salivary gland.

with bacteria. Before the sutures were passed they were soaked in a suspension of bacteria, these being actively hemolytic staphylococci and streptococci, the former having been isolated from the blood of a case of staphylococcus septicemia and the latter from a case of acute pharyngitis.

Intratracheal ether anesthesia was used in each operation, and, in order to secure conditions well removed from the ideal, no

Fig. 2.—Tonsil lymph node, showing globules of pigment along cords. This specimen was taken from an animal which was killed one-half hour after the tonsil region had been injected with a turpentine-ether solution of Berlin blue. This was done to determine and identify the lymph node, which is next in series with the tonsil. It demonstrates how rapidly absorption from the tonsil into the lymph node takes place. The small globules of dye can be seen as innumerable black spots following the distribution of the lymphatic cords. No tissue stain has been used in this section.

antiseptic cleansing of the throat was done before operation and no sterilizing of hands, instruments or gauze was attempted. Two of the animals were killed at the end of two days and the third

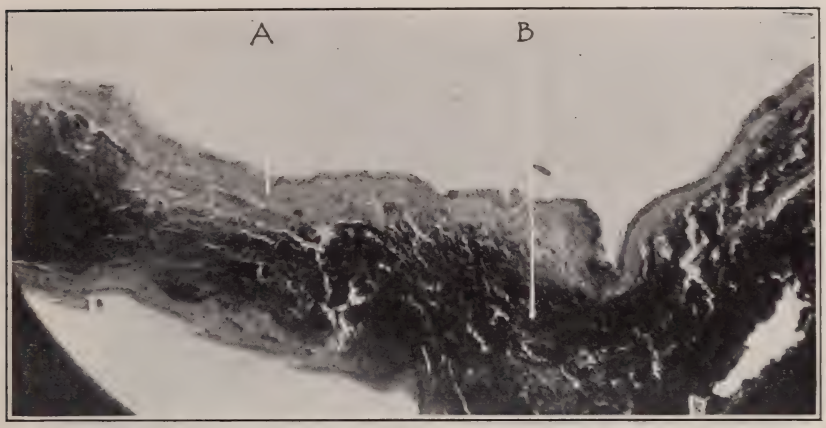


FIG. 3.—Tonsil pillar, showing submucous tissue distended with recent blood clot, two days after operation. This photograph illustrates one of the results of operative trauma on the tissue immediately adjacent to the tonsil. *A*, faucial mucosa and submucosa; *B*, muscular and fibrous tissue infiltrated with blood.

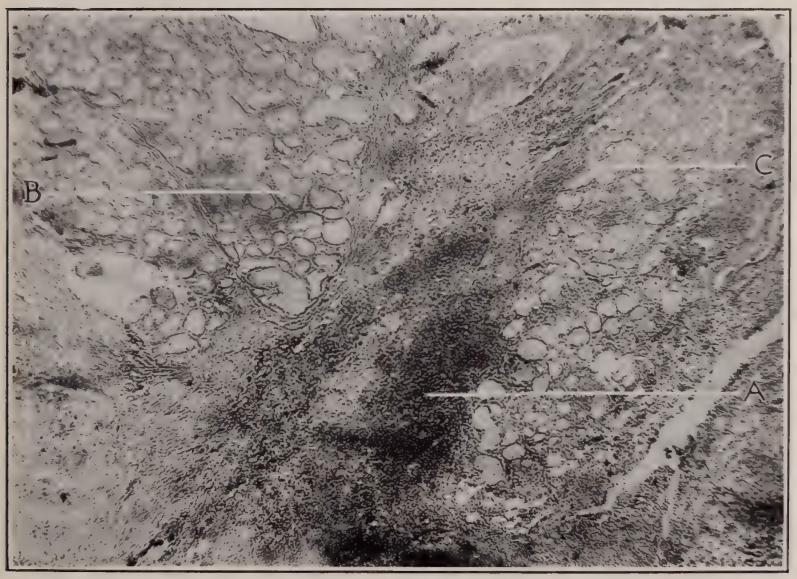


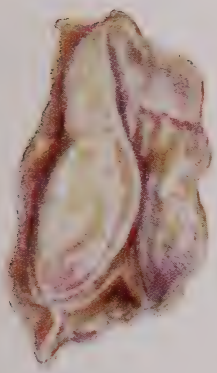
FIG. 4.—Salivary gland adjoining operation area. This specimen demonstrates hemorrhage into the gland as seen two days after operation. The hemorrhage shows as the dark structureless areas in the center and spreading out between the tubules. The pressure of the hemorrhage and the deprivation of blood supply have caused the gland cells to degenerate and the acini to lose identity; incidentally all parts lose contrast. *A*, hemorrhage; *B*, degenerating salivary gland, but some identity retained; *C*, more seriously destroyed salivary gland.

after a lapse of four days. Various blocks of tissue were removed and studied histologically as well as for bacteria.

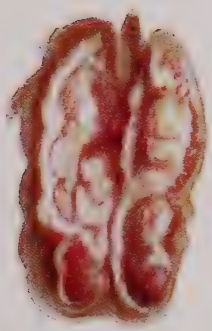
This part of the work was done in the Laboratory of Surgical Research of the University of Pennsylvania, and we are greatly indebted to Dr. J. Edwin Sweet for his helpful coöperation.

FIG. 5.—Operation field near salivary gland two days after operation. This picture shows hemorrhage and dense polynuclear and round-cell infiltration into the muscle tissue under the operation site and into the near-by salivary gland. *A*, raw operation field; *B*, muscle tissue with marked inflammatory cellular invasion; *C*, salivary gland with similar cellular invasion.

RESULTS OF PRESENT INVESTIGATIONS. Study of our material, both gross and microscopical, has elicited the following facts:



(a)



(b)

FIG. 6

The Tonsil. The position of the tonsil in a narrow recess between and almost covered by the pillars, as depicted by Sisson,[15] was confirmed.

The Tonsil Lymph Node. The large lymph node lying under the occipitomandibularis muscle lateral to the pharyngeal wall and anterior to the sternomastoid was (Fig. 1) identified as the

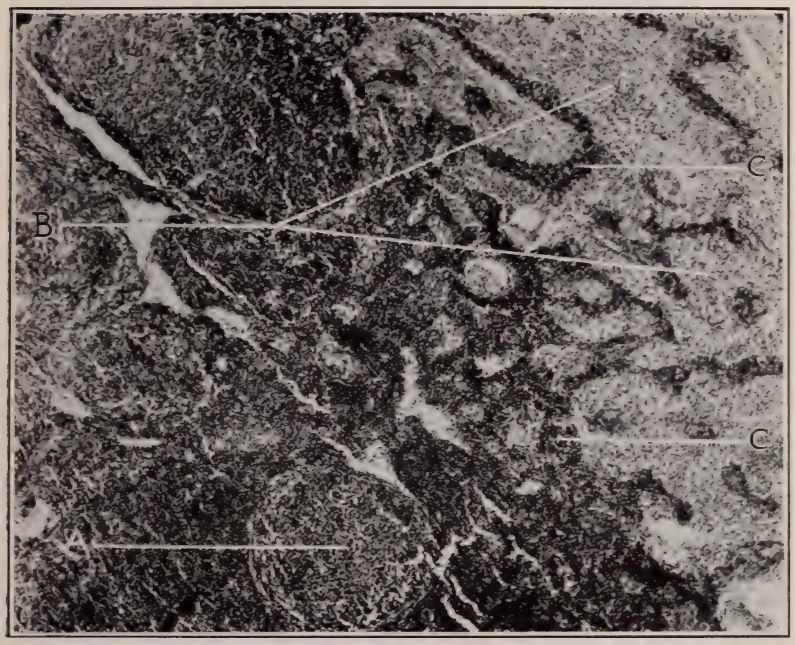


FIG. 7.—Section of the tonsil lymph node depicted in Fig. 6, *a*. The tonsil on this side of the body had not been removed. Specimen was removed two days after operation. While this node shows moderate activity in the follicles, all proportions are correct and the sinuses and cords are practically normal. *A*, follicle; *B*, sinuses; *C*, small normal cords.

drainage node next in series to the tonsil. Turpentine-ether solution of Berlin blue injected into the tonsil was found to have drained rapidly (well under one-half hour) into this node. Lymph vessels were found, visible to the naked eye, connecting the lateral pharyngeal wall with this node. Dissection (Fig. 2) and microscopy of the tonsil revealed globules of the stain in the sinuses

and along the cords of the tonsil, and also in the loose peritonsillar connective tissue. In the lymph node (to be called hereinafter the "tonsil lymph node") it was found to some extent under the capsule, but mainly along the cords within the node.

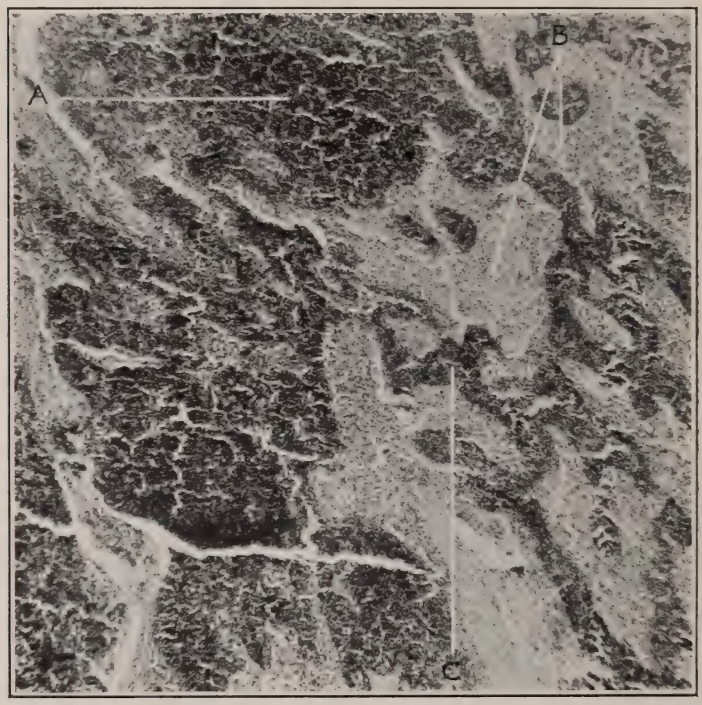


Fig. 8.—Section of the tonsil lymph node depicted in Fig. 6, *b*. The tonsil on this side of the body had been removed. Specimen removed two days after operation. The lymphoid tissue is very active and the markings are obscure, there being for instance no germ center in the two follicles in the picture. The sinuses are distended with blood, and the cords are wider than in Fig. 9. *A*, follicle showing activity; *B*, blood-filled lymph sinuses; *C*, cords.

Local Postoperative Effects of a Purely Traumatic Nature. The first effect of tonsillectomy, that of trauma, was found in our material principally as hemorrhage. There was a pouring out of blood (Fig. 3) not only into the tissues immediately subjacent to the operative field, but also in those lateral to it, under the intact epithelium, and extending well beyond the pillars of the

tonsil (Fig. 4). In addition, hemorrhage was found in the adjacent salivary gland with resultant degeneration from pressure and impaired nutrition. Further reaction to the operative procedure was found in the form of round-cell and polynuclear infiltration, followed later by fibrino-cellular exudation, the latter varying with the infection (Fig. 5). This cellular infiltration reached the lateral and deep tissues, and among its sinister possibilities is destruction of these tissues, as happened to the salivary gland in these experiments.

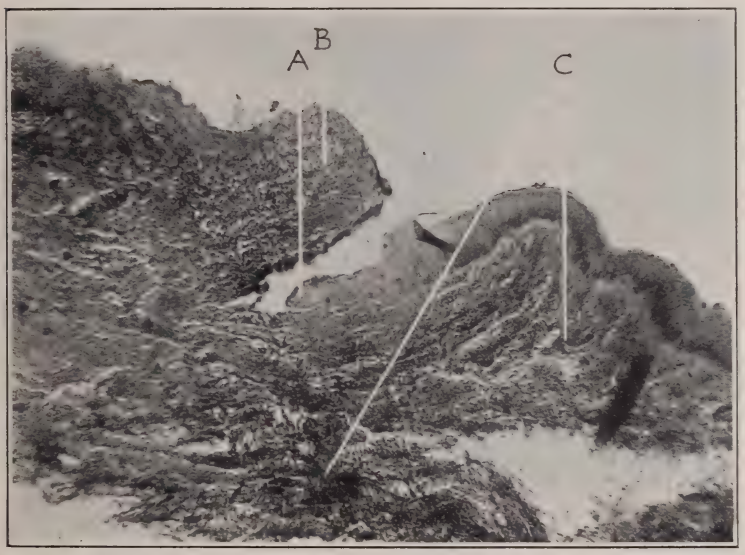


Fig. 9.—Edge of the operation site, showing postoperative exudate overlying the edge of the epithelium two days after operation. *A*, end of the epithelium at the edge of the operative field; *B*, edge of the postoperative exudate overlying, but here artificially separated from, the epithelium; *C*, blood under the epithelium at the site of the operated area.

Over the operation field a fibrino-cellular sheet was found, the superficial layers of which are progressively cast off, just as false (Fig. 10) membrane loosens, until the healing area beneath it is completely exposed.

The Effect of Tonsillectomy upon the Tonsil Lymph Node without any Attempt Having been Made to Infect the Operation Wound.

Two days after operation blood was present in the tonsil lymph node. The node was swollen and, when sectioned, was found to be so full of fluid (Fig. 6) that the cut surface was wet and greatly congested. The node on the unoperated side was pale pink or yellowish and less wet.

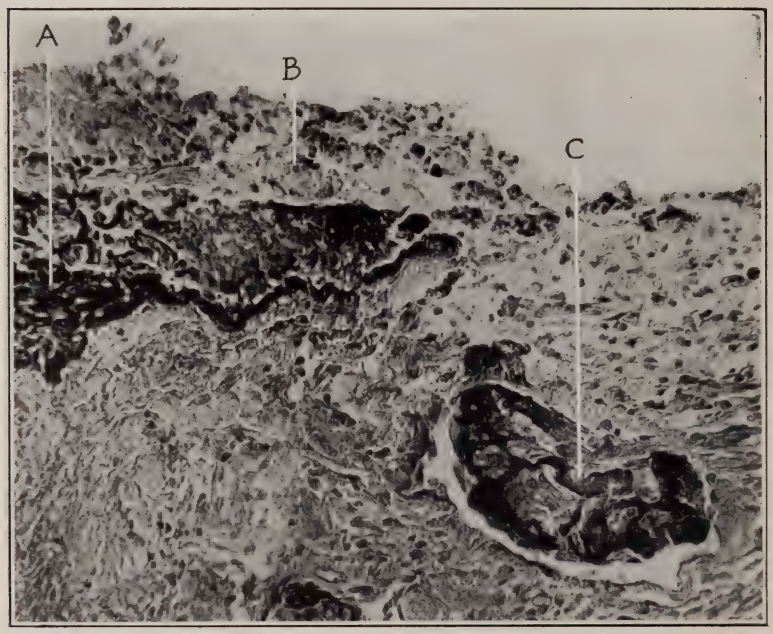


Fig. 10.—A portion of the operation field four days after operation, with the tissues stained for fibrin. The dark parts are fibrin. On the surface is an exudate consisting of two layers, a superficial light and a deep dark one. The former represents the actively degenerating part of the exudate, while the latter is the firm fibrinous exudate itself. The latter lies upon the healing operation base, and will itself later degenerate and be cast off. A well-formed thrombus lies in a vein just below operation area. *A*, fibrinous exudate; *B*, degenerated part of the exudate; *C*, thrombus.

Under microscopical study the node on the operated side was seen (Fig. 8) to be full of blood cells and their fragments, the sinuses especially being widely dilated. The lymphoid tissue itself was active in proliferation, but the follicular areas were not emphasized, there being instead a diffuse lymphocyte increase of all parts.

On the unoperated side the node was found to be active in its (Fig. 7) follicular areas, but otherwise normal.

The Peritonsillar Results of Tonsillectomy which are not Purely Traumatic and which were Found when the Wound had been Artificially Infected. At the area of operation there was found, two days after operation, a well-developed fibrino-cellular exudate

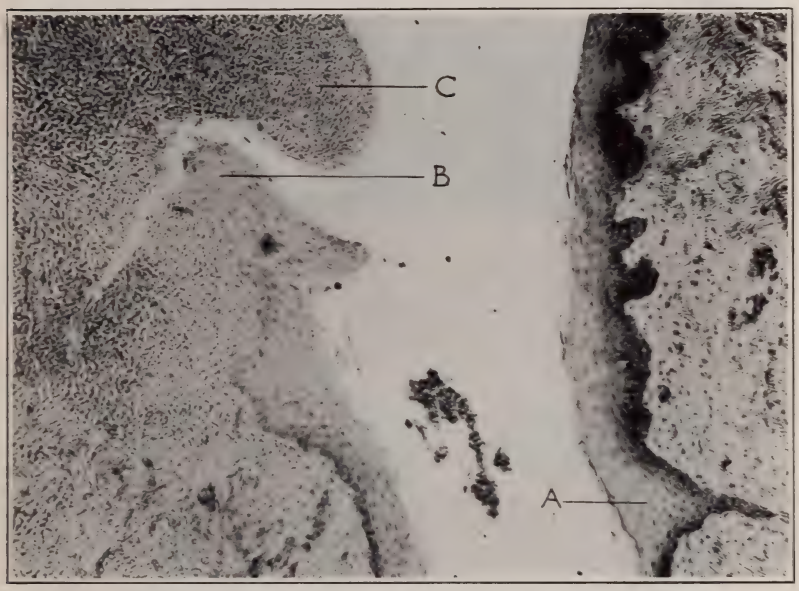


FIG. 11.—Tissue at the edge of the operation area, two days after operation. This figure shows the two lips of the wound, the one with undisturbed epithelium, the other at the cut edge where the epithelium is trying to spread but meets the exudate at the operation site. The epithelium is attempting to cover the raw surface but the exudate overlaps the proliferating surface cells. *A*, normal epithelium; *B*, inflamed epithelium at the edge of the wound; *C*, edge of the exudate overlying *B*.

(Fig. 9). This not only covered the raw surface, but also extended laterally to overlie the edge of the adjacent epithelium. This exudate formed a barrier (Fig. 12) to the extending of the epithelium over the raw surface. In addition, when infection was present the stratified layers of epithelium were seen to be thickly beset with polynuclears (Fig. 13) and in a state of active inflammation, this decreasing as the distance from the operation site increased

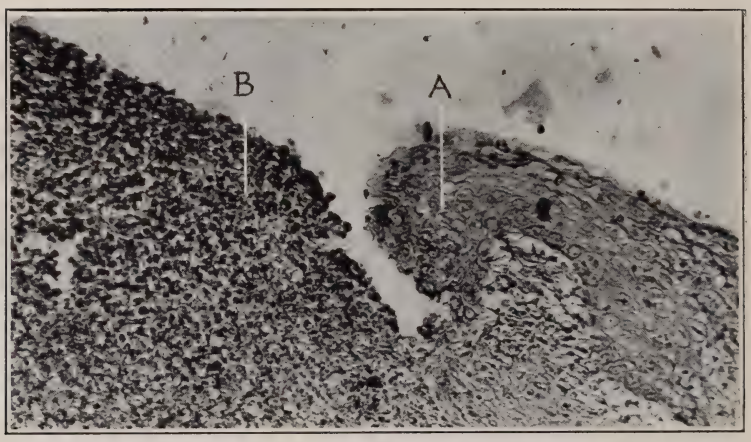


Fig. 12.—Edge of the tonsillectomy area, two days after operation. This figure shows the edge of the epithelium unable to make headway against the infected actively inflamed base of the operation site. *A*, edge of the epithelium; *B*, inflamed tissue showing the fibrino-cellular exudate developed at the operation site.

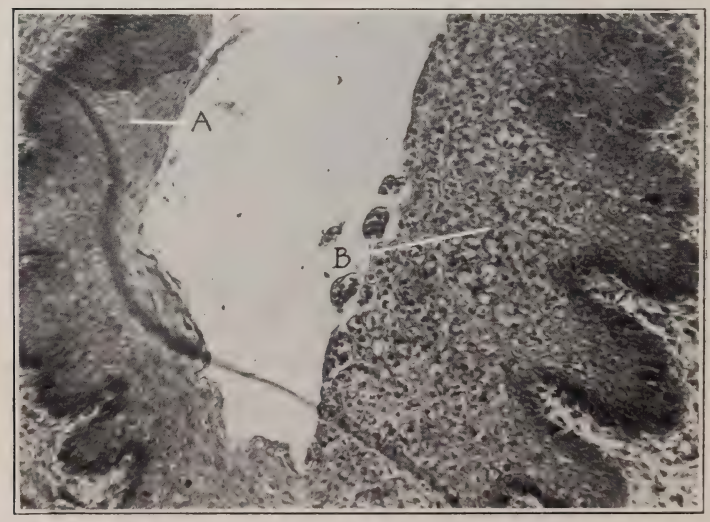


Fig. 13.—Fold in the mucous membrane adjoining the edge of the operation area, two days after operation. Normal epithelium at a distance from the wound. Actively inflamed epithelium adjoining the edge of the wound. *A*, normal epithelium distant from the wound; *B*, epithelium near the wound, showing active inflammation with many polymorphonuclears.

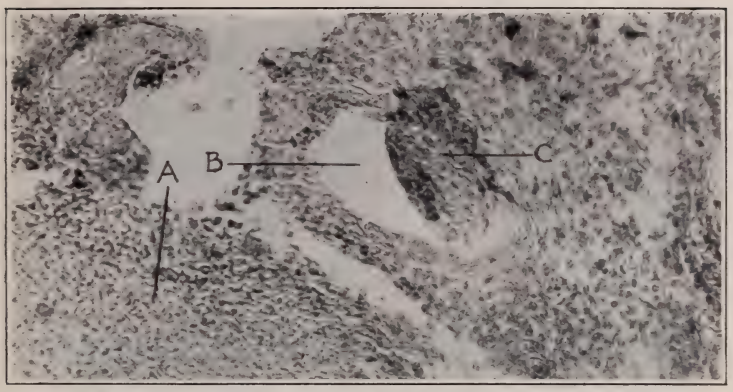


FIG. 14.—Base of operation site, two days after operation. This specimen shows dense infiltration, edema and a thrombosed vein. *A*, dense infiltration of round and polynuclear cells and much edema; *B*, thrombosed vein; *C*, thrombus artificially detached.

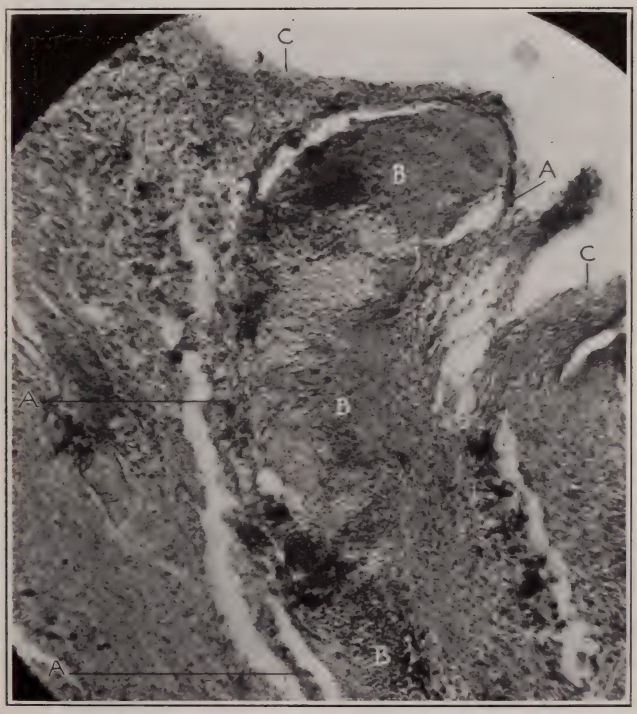


FIG. 15.—Thrombus in a vein just under the operation site, two days after operation. At several points the thrombus will be seen to be quite free from the lining of the vein, but at most of its periphery it will be seen to be closely attached. *A,A,A*, wall of vein; *B,B,B*, thrombus; *C,C*, raw operation field.

(Fig. 10). The exudate lay upon a tissue base characterized by a reactive cellular inflammation.

A noteworthy finding in this inflamed part was the congestion of all varieties of vessels and the frequency of thrombi, both near the surface and deep in the tissues (Figs. 14 and 15). These thrombi were attached to the vessel wall and were well developed by the end of the second day. No signs of softening were observed in any of them.

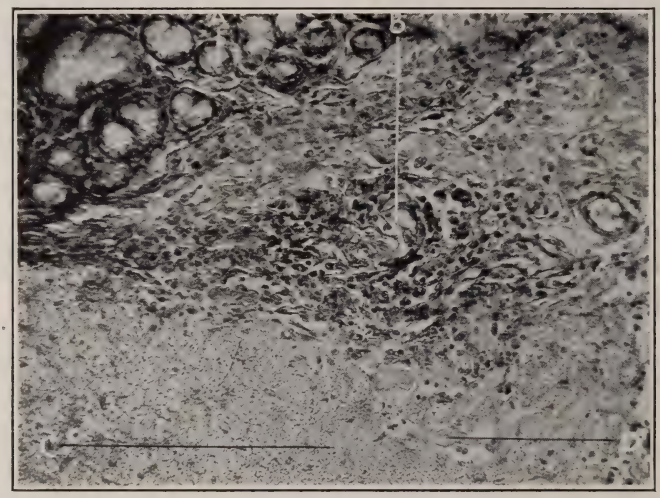


Fig. 16.—Deep tissue beneath the operation site, two days after operation. This specimen shows vacuolated salivary gland cells, and separation with destruction of a small piece of salivary gland by cellular infiltration. It also shows edema and degeneration of a large area of muscle, small strands of which are recognizable. *A*, salivary gland with vacuolated cells; *B*, isolated island of salivary gland surrounded by cells and edema; *C*, degenerated muscle, strand remnants of which are seen at *D*.

Study of the deeper tissues of the tonsillectomy area showed the inflammation to be similar to that near the surface, including a feature already noted, viz., the involvement of the salivary gland (Fig. 16). In addition the fibrino-cellular exudate was seen to extend well into the muscle tissue (Fig. 15), and many small thrombotic veins were here encountered.

The Local Effect of Sutures. The suture tracks were found to be surrounded by a mild fibrino-cellular reaction, highly cellular

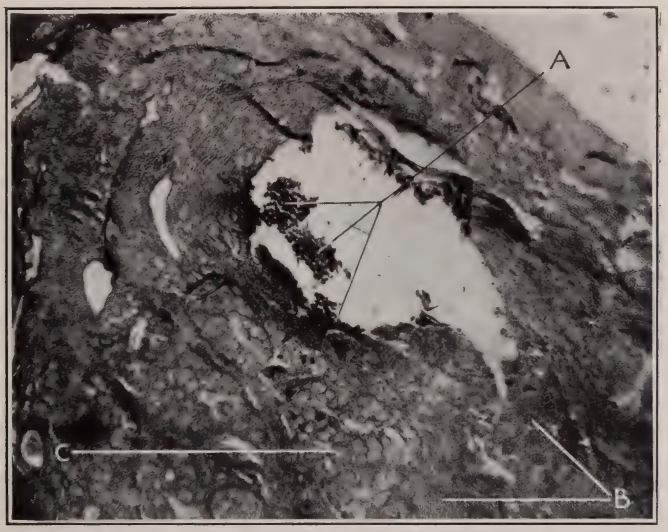


FIG. 17.—Silk stitch and adjacent area, under low magnification, four days after operation. Most of the stitch fibers have fallen out of the section. This picture shows a healthy reactive area around the stitch, with some degeneration of muscle and salivary gland tissue. *A*, fibers of the suture; *B*, degenerated muscle; *C*, degenerated salivary gland.

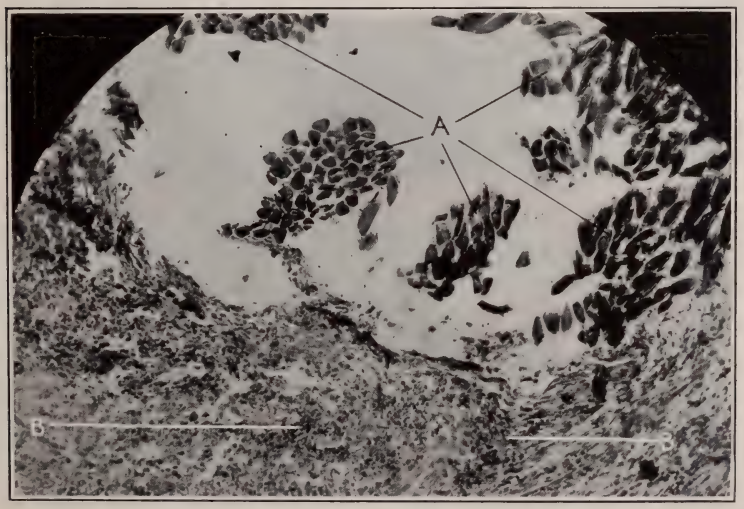


FIG. 18.—This picture shows the stitch and its adjacent tissue, under high magnification, four days after operation. *A*, strands of the stitch; *B,B*, healthy reaction to the stitch, some round-cell infiltration in small groups, but no abscesses.

at the end of two days (Fig. 17), and showed signs of healing at the end of four days. The tissue caught by the stitch was compressed and degenerated, particularly the muscle and salivary gland tissue (Fig. 18). Small patulous capillaries were found in the healing area, but the large veins usually were thrombotic.

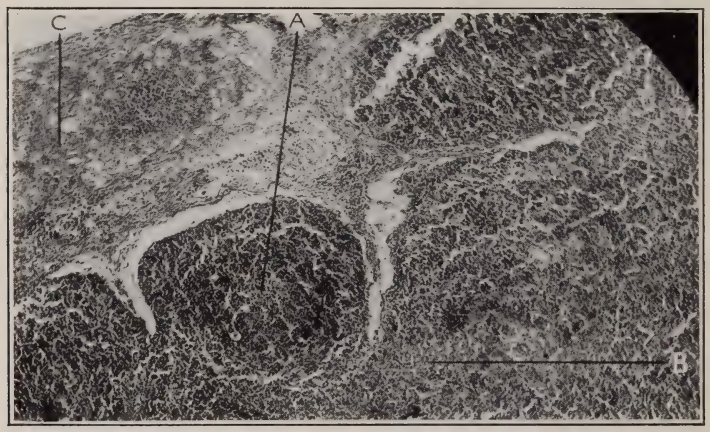


Fig. 19.—Tonsil lymph node, two days after operation, on the side of which infected stitches were used to close the wound of tonsillectomy. This node shows a very active hyperplasia, as indicated by the size and solidity of the follicles, a degree of compactness of the intervening lymphoid tissue great enough to obliterate the sinuses. In addition, there are congestion of the capsular vessels and distention of the lymph ducts. In certain of the capsular lymph channels some swelling and degeneration of the lining were found. Bacteria were found in these lymph ducts. *A*, hyperplastic follicle; *B*, solid cordal area without visible sinuses; *C*, swollen, congested and edematous capsule.

The Tonsil Lymph Node after Operation Plus Artificial Infection of the Operation Wound. The tonsil nodes after an infected operation were very large, soft, wet and irregularly congested, especially at the capsule (Fig. 19). Under magnification a very marked hyperplasia was found, differing greatly from conditions depicted in Figs. 9 and 10. The follicles showed large germ centers and the cords were crowded with mononuclears. The sinuses were compressed almost to extinction by the lymphoid increase or were filled with large and small mononuclears (Fig. 21), as well as with endothelioid and polynuclear cells. This last change was best seen near the hylum of the node. In addition, the increased

size of the lymph vessels was quite notable. They were greatly dilated and were almost completely filled with a hyaline postmortem lymph clot (Fig. 20), this being especially notable in the capsule. Bacteria were found in or near the lining of these vessels.

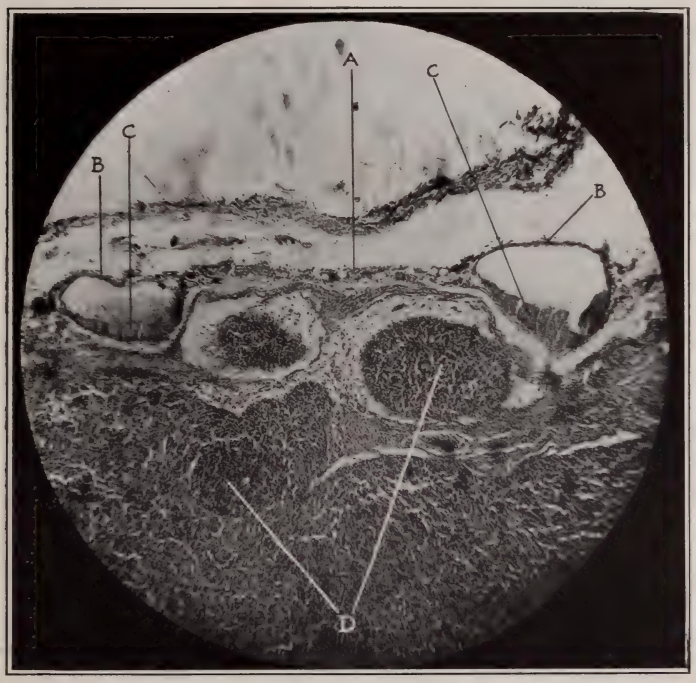


Fig. 20.—The same lymph node as that from which section shown in Figs. 19 and 21 were taken. It shows dilated lymph vessels with postmortem clots. A small group of bacteria was found in one of these clots near the lining of the vessel. Solid hyperplastic follicles of the node lie under the capsule. *A*, capsule of the lymph node; *B,B*, lymph vessels; *C,C*, coagula; *D*, follicle.

It should be stated in this connection that a superior mediastinal lymph node of one animal showed a similar hyperplasia. There were present also dilated lymph vessels, small hemorrhages and very marked degeneration of the follicular centers. Bacteria were not surely found.

Bacterial Distribution. Bacteria in those cases which were artificially infected were found with ease in the stitches and in the

area immediately surrounding them (Fig. 22). As distance from the stitch region increased the microörganisms became fewer and

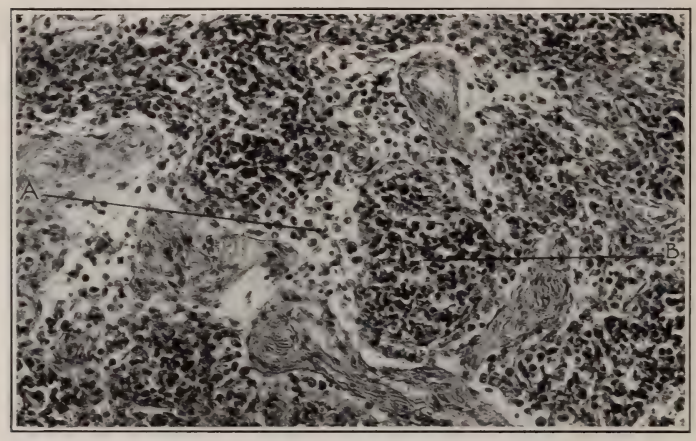


FIG. 21.—The same lymph node as that from which sections shown in Figs. 19 and 20 were taken. Central area of the node under high magnification. This specimen shows the cellular richness of the central sinuses. Numerous lymphoid cells of all varieties, polymorphonuclears and large mononuclears of endothelioid proportions were found in the well-crowded spaces. The cords are very thin at this point. *A*, a crowded sinus; *B*, the end of a small cord.

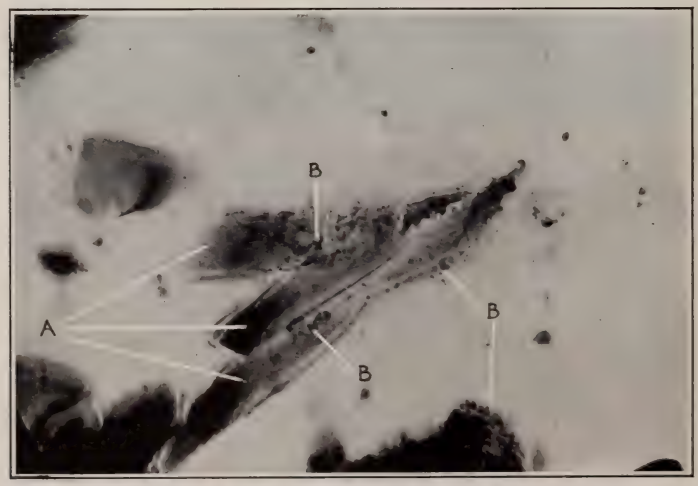


FIG. 22.—A strand of stitch with bacteria in it, four days after operation. (Oil immersion exposure.) *A*, fibers of silk; *B,B,B*, bacteria.

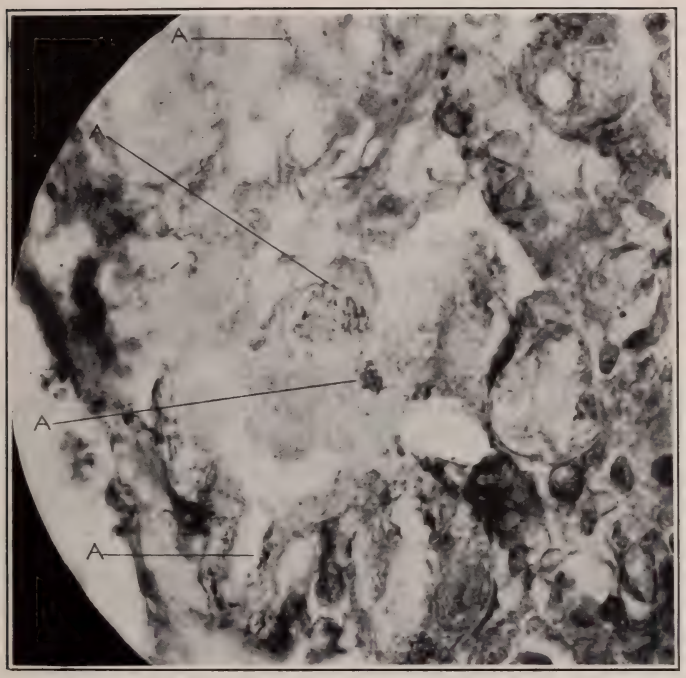


FIG. 23.—The salivary gland from animal artificially infected by stitches soaked in cultures, four days after operation. This photograph shows bacteria singly and in groups. *A,A,A,A*, bacteria.

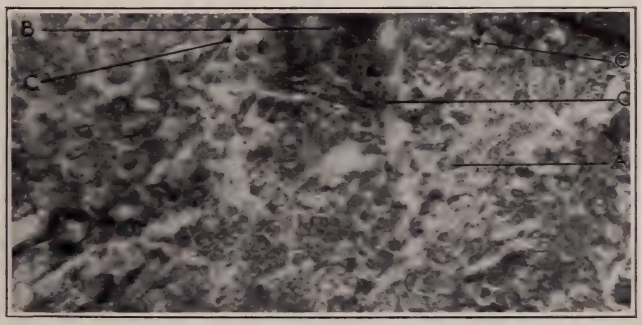


FIG. 24.—The tonsil lymph node from animal artificially infected by stitches soaked in cultures, four days after operation. This section shows bacteria on the edge of a cord and in a necrosis at the edge of a cord. *A*, cord; *B*, necrosing area; *C,C,C*, bacteria.

fewer. They were traced along one tissue split into a muscle area (Fig. 23), and also were found in a salivary gland.

In the tonsil lymph node bacteria were present in pairs and in small clumps. They were found in the dilated capsular lymph vessels (Fig. 24), in the subcapsular (the so-called "marginal") sinus and in the interior sinuses near the edges of the cords.

They were not found with unequivocal certainty in the thrombosed vessels.

Summary of Results. Injected material found its way from the tonsil to the first drainage lymph node in half an hour or less.

Tonsillectomy traumatism was manifested not only at the immediate operation site, but also in the collateral tissue in the form of hemorrhage and cellular infiltration.

The surface of the wound was covered with a fibrinous exudate, the outer layers of which were progressively cast off.

The tissue reaction at the site of operation was degenerative and fibrino-cellular, the former appearing at once, the latter being in part the result of traumatism and in part the natural successor to degeneration, and therefore the early stage of repair.

Exudate of a fibrino-cellular character was found deep in the tissues, with resultant degeneration of muscle and salivary gland tissue.

Many venous thromboses were found both near the surface and deeper.

The tonsil lymph node reacted to moderately clean operative procedures by caring for the extravasated blood and by mild hyperplasia.

To a purposely infected operation the reaction of the lymphatic tissue was very marked, so much so that the lymph current probably was obstructed.

Each attempt at artificial infection succeeded and important structures, such as muscles and glands, were found to have been invaded by the microörganisms.

Bacteria were found easily in the surface exudate and in the vicinity of infected stitches. They also were found in the salivary glands and in the tonsil lymph nodes.

When bacteria-soaked stitches were introduced the infected or at least inflamed, area was thereby made deeper, while important tissues were compressed and distorted.

While not certainly demonstrated in the thrombi near the operation wound, we believe that bacteria sure must be there. The fibrin was so dense (having taken the same stain as did the bacteria) that their definite identity was obscured.

* * * * * * * * * *

We have shown graphically what happens around a tonsillectomy wound. In a word, there are present hemorrhages, thrombi, necroses and bacteria. Hemorrhage causes rifts in the tissues, opens pathways for infection and destroys tissue. Trauma and disturbed blood supply result in necrosis. Injured bloodvessels are closed by coagula which may become infected and loosened. Bacteria are carried into the tissues by physical force or are permitted to penetrate by the opening up of tissue clefts, especially when there has been some devitalization. These are present in an area which never can be put at rest, which constantly is open to reinfection and which is subjected to a great deal of constrictor muscle action.

What might be the consequences? Should a sterile thrombus be dislodged into the blood the result would be a small sterile pulmonary infarct which, however, later may become infected and give rise to pneumonia or an abscess. Should a septic thrombus be dislodged a lung abscess would be the probable result. Should a thrombus, either sterile or septic, reach a previously diseased area of the lung, such as a tuberculous focus, the lesion might be activated.

As yet there has been no postoperative study of tonsil cases in which the lungs have been carefully watched for small lesions. Lung infarctions after tonsil operations naturally would be small on account of the size of the peritonsillar veins, and it is perfectly possible that many a lung infarct caused by a non-septic embolus has escaped notice. *We would like to emphasize the following point:* One of the early signs of pulmonary infarction is the spitting of

bright red blood, and it is quite possible that at times some of the blood-spitting in the early days following tonsillectomy is due to an overlooked infarction and not to oozing from the operation field. In our clinical work it might be well to keep this in mind and let this sign be a signal to examine the larynx and trachea as well as the tonsil fossæ, and also to have the chest examined and roentgen-rayed.

Conclusions. 1. The peritonsillar region as depicted in our specimens constitutes a postoperative field studded with thrombi, which may be either sterile or septic.

2. We believe that as trauma, sepsis and muscular action are the principal factors in the formation and dislodgment of thrombi, the actively muscular pharynx is a particularly favorable region for the dislodgment of such thrombi into the superior caval circulation.

3. We believe that many cases of pulmonary embolism escape notice on account of their small size, their sterile character and their prompt resolution.

4. It goes without saying that the performance of tonsil operations should be attended with every possible surgical device for promoting asepsis. We suggest, in the furtherance of this principle, that the routine use of needle-carried sutures for tying bleeding vessels be abolished and that surface ties be used instead.[16] The former are never surgically clean and we believe that their use carries with it possibilities of infection of at least the tissues adjacent to them.

5. While realizing that posttonsillectomy lung complications may at times be of inspiratory origin, we believe that such origin is not as common as has been thought.

6. While the infected condition of the lymphatic tissue found in our study must cause consideration to be given to a lymphatic origin for lung infections, we consider such origin to be rare.

7. Finally, it is possible that, in view of such studies as the present one, ideas in regard to the capsule of the tonsil may undergo a change. Instead of aiming to remove it, we may decide that it forms too good a bulwark to the peritonsillar tissues to allow of

its being taken away. And we believe it to be entirely possible that some day the operation, called by Makuen[17] "intracapsular tonsillectomy," will be so demanded by the highest ideals of safety that in a perfected form it will be the operation performed by all of us.

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DISCUSSION

Dr. Eugene L. Opie: I have listened to this paper of Dr. Fetterolf and Dr. Fox with especial interest because I have heretofore strongly inclined to the inhalation view of posttonsillar pneumonia and abscess. I think Dr. Fetterolf and Dr. Fox have very clearly demonstrated the embolic origin of some of these lesions, yet I recognize that it is very difficult to decide the relative frequency of embolic and bronchiogenic infection. In human infection, as distinguished from the experimental, the subject is very closely associated with the hemolytic streptococcus, which

has its natural habitat in the tonsil and is unquestionably the specific cause of follicluar tonsillitis. This organism was found by Dr. H. A. Bell, at Washington University, in at least 70 per cent of tonsils which were removed at operation. This microörganism is one especially adapted to enter lymphatics, to bring about thrombosis of bloodvessels and to cause suppuration of the thrombi, with dissemination of the microörganism by the blood.

It may be worth while to recite some of the arguments advanced by those who have maintained the bronchiogenic origin of postoperative pneumonia: Bacteria doubtless can pass even from the normal mouth into the lower respiratory passages. Experiments with readily recognizable organisms, such as Bacillus prodigiosus, show that when introduced into the nasopharynx and mouth it may appear in the lower respiratory passages. Indeed, very recently Mullin and Ryder have found that India ink introduced in the nasal passages of the rabbit placed on its back makes its way in considerably quantity into the bronchi. There appears to be little doubt that microörganisms brought to the nasopharynx with the inhaled air may penetrate into the lower respiratory passages. With tonsillectomy there are some conditions which specially favor the penetration of microörganisms, notably hemolytic streptococcus, into the lung. Studies on the bacteria of the mouth have shown that the flora is very constant. The hemolytic streptococcus does not stay in the mouth unless it already has a lodgment in the crypts of the tonsils. With the flushing of the mouth by saliva, and especially by currents of mucus closely attached to the mucous surfaces, microörganisms are carried into the esophagus, as Bloomfield has shown. Following tonsillectomy there is impaired deglutition, and often a constant source of virulent microörganism, thus affording favorable opportunity for penetration into the lower respiratory passages.

It is noteworthy that hemolytic streptococcus is the usual cause of lung abscess following tonsillectomy. The same microörganism is the most common cause of the lung abscess following influenza, and here it appears evident that the destructive changes in the bronchi associated with the bronchitis of influenza facilitate the entrance of hemolytic streptococcus into the bronchial passages, the origin of the abscess thus being bronchiogenic. It is probable that streptococcus may be transported from the tonsils to the lungs both by the bronchi and by the blood stream. Dr. Fetterolf and Dr. Fox have made it very clear that every precaution must be taken to prevent embolic pneumonia.

Dr. Chevalier Jackson: I have not operated upon the tonsil for fifteen years, but I have recently recalled cases which I now believe were

posttonsillectomic pulmonary abscesses. Curiously enough, the region of our country in which the greatest number of posttonsillectomic pulmonary complications occurred in Moore's statistics is the same region in which the tonsils are operated upon in the upright position under general anesthesia. Dr. Moore and all other bronchoscopists have abundant evidence of how easily safety-pins, tacks, nails, horses, dogs, goats, opera glasses—toys, of course—peanut kernels and in fact almost any and every small object can be aspirated down into the lungs. But we never see, except in peanut kernels in children, the very rapid destruction of lung tissue shown in the specimen just exhibited by Dr. Crawford. So far, we have not found any tonsillar tissue or clots in posttonsillectomic cases bronchoscoped, but none was done in the incipiency of the suppuration. As corroboration of the protective activity of the lung spoken of by Drs. Fetterolf and Fox, I might mention the work done by Dr. Ernest Willetts fifteen or more years ago. By smears and cultures from uncontaminated specimens that we removed bronchoscopically from deep down in the bronchi he found the secretions sterile. This was so even in a number of cases in which a metallic foreign body had recently been aspirated. The foreign body must have been contaminated when it was aspirated, because it necessarily went through the mouth; yet the protective activity of the deeper bronchi had in a few days cleansed it of live organisms. Dr. Stillman's experiments, showing that aspirated pneumococci are destroyed in a few days whereas streptococci cause suppuration in the lungs of mice, taken in connection with Dr. H. H. Lott's work on the significance of streptococci as differentiated from other organisms in tonsillar focal infection, seem to me of fundamental importance in the consideration of posttonsillectomic pulmonary abscess by whatever route the infection may reach the lung. Dr. Moore and other bronchoscopists have abundant demonstrations of the ease with which almost anything can slip down the windpipe into the lungs, and Dr. Moore has demonstrate cases that were undoubtedly aspiratory in origin. Dr. Fetterolf and Dr. Fox have demonstrated how direct is the hematogenous route and have presented indisputable evidence that cases of abscess occur this way. Undoubtedly these cases occur both ways; it remains only to determine the percentage occurring each way. Another thing proven by Dr. Fetterolf, Dr. Fox and Dr. Moore is that pulmonary abscess is a complication to be reckoned with in advising tonsillectomy, especially the radical operation in adults.

Dr. John B. Roberts: A statement was just made by a previous speaker that there is no surgery worse than that used in tonsillectomy. Those of us general surgeons not skilled in this operation at all have seen

things which seemed to us to be almost brutal. I do not certainly know whether or not it is due to the necessities of the procedure or to the operation being undertaken by men unfamiliar with surgical technic, but I believe the latter is the truth.

Fifteen minutes ago I said of this communication of Dr. Fetterolf and Dr. Fox, that these gentlmen have probably brought here tonight the most important and the best piece of work they have ever done. In etherization of some cases of tonsillectomy I have seen bad technic in several ways. I do not say surgeons, as a rule, would do any better, but that they scarcely could do worse. Many surgeons perhaps could not do as well, because they do not get much chance to do this operation. What I have observed, say for sixteen years, is that many laryngological operators on the tonsils are too much afraid of hemorrhage. I have seen throat specialists use apparatus in a little child's throat powerful enough to crush off one of its fingers with its pressure obtained through a revolving lever. This diabolical tool is used lest a little artery or two should spurt when the knife or scissors is cutting. A surgeon would cut away, or dissect out, the tonsil from its bed, pinch the two or three small bleeding arteries with the pointed end of one or two hemostatic forceps, leave them in position while washing out the throat and mouth, then give each hemostat a couple of twists and remove it. This is probably a better method than a puckering stitch, making a pocket to imprison blood and secreta.

Should this not arrest the flow of blood, he would allow the forceps to remain for a few hours with the handles protruding from the mouth. Should the bleeding be more difficult to stop than usual, an old-fashioned pointed hook, called a tenaculum, could be used effectively. It should catch up a small portion of tissue near the open end of the bleeding vessel, twist the soft tissues by rotating the flat handle of the tenaculum. The surgeon then closes the teeth upon it and holds the mouth shut for an hour or two, with a nurse or attendant sitting by the child to allow its mouth to be opened if there happens a tendency to vomit.

A better term for tonsillectomy is amygdalectomy, just as one says nephrectomy instead of renectomy and proctitis instead of rectitis. It does not appear a difficult operation, even so far as bleeding is concerned, to a surgeon accustomed to operations in the cavity of the mouth.

Finally, I have never been convinced of the wisdom of removing normal or nearly normal tonsils as a preventive against general infection. I prefer to keep a child free from oral infections by keeping its teeth clean, and dirty fingers and nails out of its mouth.

Dr. Hobart A. Hare: I would like to emphasize the point brought up by the previous speakers concerning the matter of statistics of post-

operative tonsillectomy. As Dr. Stengel said, every general medical man who has a private or hospital practice of any size sees cases of pulmonary disorder he cannot explain, until by some accident, if you choose, he learns that six, eight or ten weeks previously the tonsils had been operated upon; and I have seen cases in which it was known that the tonsils had been operated upon, and yet the time seemed to be so widely separated that neither tonsillotomist nor the physician thought there was any relationship of cause and effect. There are some general practitioners bold enough to remove tonsils; indeed, I understand a great many general practitioners are bold enough to do this; but I think tonsillotomy should be considered a definitely major surgical operation, not to be done by anyone except after the greatest possible training and skill. I think one of the reasons actuating novices is that in their student days they were taught the old guillotine operation, of shearing off part of the tonsil and leaving the rest behind. The statistics of the Mayo Clinic would seem to mean that there have been operative recoveries in the sense that the patients no longer complained of their throat and left the institution in a short time. What Dr. Roberts said about taking out healthy tonsils is akin to taking out the healthy appendix in the old days; it is also akin to taking out healthy ovaries. I recall the story of a gynecologist in a neighboring city who did an abdominal operation on the operative and then took out the healthy ovaries, the patient being in a lowly walk of life, and on being asked why he did it, he said, "Well a person in this walk of life is likely to fall from grace, and I thought it was just as well to remove her ovaries on general principles." The general removal of tonsils approaches this point of view.

(I think, perhaps, Dr. Stengel would be a little more interested in the etiology of these suppurations if he were doing nose or throat operations or bronchoscopy.)

Dr. W. F. Moore: The relative importance of these two modes of infection, that is blood-stream infection or aspiration, is, I believe going to be of great interest to us and greater help to us in the future, because if these infections are blood-borne, it may make a difference in the time at which these cases should be attacked bronchoscopically. That is, if they begin as infarcts there is not the primary involvement of the bronchus or bronchi, which carries infection to the infected area, that there would be if due to inspiratory origin. For this reason, the men doing bronchoscopy have been interested in the etiology of these lung suppurations. Now, these cases collected around Boston that Dr. Jackson called your attention to comprise 23 of 40 cases which followed tonsillectomy in a semirecumbent or upright position. Incidentally, 40 local tonsillec-

tomy cases followed by lung abscess was a large percentage in 202 cases which were collected after tonsillectomy had been done. So you can see from this that with this ratio (40 in 202), and with local tonsillectomy being done less frequently than with general anesthesia, that it does follow local tonsillectomies in a large percentage of cases. Furthermore, these cases from around Boston were done in the upright position or semi-recumbent position, under ether anesthesia, we attributed the subsequent lung suppuration in these cases to the fact that gravity probably played a large part in conveying the infection to the lung. The inhibition of the superior laryngeal nerve due to the blocking of the local anesthesia must be remembered where local anesthesia is used. Dr. Opie referred to the bacterial studies in these cases. Our studies last year were done in a rather unsatisfactory manner, inasmuch as the infected material which was taken from the bronchoscope by swab, or specially devised tubes by suction, was some time in being sent to the laboratory. The results were rather uniform. We found infections were due either from staphylococcus, streptococci or catarrhalis. It was rather interesting that streptococci were last to disappear after bronchoscopic treatments, including the aspiration of infected area and washing out with special solution. Lately, Dr. George R. Moffitt, of Harrisburg, has been doing that work for us and the whole bacterial picture has changed. Due to his work, we have found that the hemolytic streptococci, pneumococci, staphylococci, B. influenza, etc., have been found both intra- and extracellularly. Another thing has been noted, and that is the swab specimens taken through the bronchoscope developed different bacteria from specimens taken from the aspirated area. We hope later to have something different to present on this, but if we can link up the bacterial side from recent local conditions in the tonsils themselves, I think we can more definitely determine later on how infection is spread in a large majority of cases.

Dr. Elmer H. Funk: A point of clinical value brought out by Dr. Fetterolf and Dr. Fox was that they mentioned the occurrence of hemorrhage early in these cases as perhaps slight evidence of infarct. Dr. Moore called my attention to this.

Dr. Alfred Stengel: Familiarity breeds contempt and, I presume, it is on this account that we have not, perhaps, taken seriously enough the possibility of abscess of the lung as a sequel of tonsillectomy, for the fact of its occasional and not very rare occurrence has been sufficiently emphasized to make it a matter of common knowledge. But one rarely hears of this possible mishap as ground for hesitation in resorting to operaation, while hemorrhage and other possibilities are, perhaps in comparison,

overemphasized. I was much interested in learning, on a visit to another city some months ago, that tonsillectomies had been performed in some 10,000 school children, under the inspiration of a layman much interested in philanthropic work, and had been performed in this wholesale number in improvised operating-rooms in schools, opera houses and other similar buildings in addition to the available hospitals without a single post-operative lung abscess; but, I confess, I was a little skeptical. One of the things that we have learned regarding the incidence of this condition is the fact that an interval of as much as several weeks may elapse between the operation and the pulmonary sequel, and it is easily possible that in the case of an operation such as this, in which the patient is frequently discharged and perhaps lost sight of after a week or little more, the subsequent developments remain unknown to the operator. Incidentally, I wish to suggest that this very matter of the long interval may be somewhat urged as an argument in favor of the embolic as opposed to the aspiratory origin of the pulmonary disease. It seems more probable that thrombi would break up and occasion embolism two or three weeks after an operation than that insufflated materials from the throat of the anesthetized patient would require so long a time in setting up pulmonary disease. Foreign bodies drawn down into the bronchi may, as Dr. Jackson has shown, remain without evident symptoms or results for a considerable time, but the case is surely somewhat different when we are dealing with the aspiration of blood, mucus and, without doubt, abundance of bacterial contamination from the throat of the patient undergoing tonsillectomy. In my own clinic, not a few cases of lung abscess have been encountered in which the development of the condition had been regarded as obscure, and remained so until a careful history discovered the fact that there was a probable immediate connection between a tonsil operation and the pulmonary disease. I cannot but suspect that pulmonary accidents and sequels are considerably more frequent than the operators appreciate, or than any of us at present recognize, and it is not too much to say that this possibility has not been sufficiently weighed in the scale on the side of avoidance of an operation the urgent necessity of which is not evident. Surgery is never a particularly agreeable enterprise for the patient, and often is, in spite of all modern advances, pretty crude business. No branch of it has seemed to me more crude than operations on the tonsils and adenoids. We cannot lay any special blame for this on the operators, for, as Dr. Fetterolf has pointed out, the nature of the field of operation is such that clean technic is difficult; but this very fact indicates the importance of such studies as that of Drs. Fox and Fetterolf, which we have just heard. Improvements in technic may be expected to follow a complete knowledge of the factors. That there is extensive local infection about the site of operation is clear to anyone who has

examined the throats of patients after such operations; that systemic infection is no more frequent than it is recognized to be, may, perhaps, be due to the open nature of the operative site and possibly to inhibitive influences of saliva or mouth secretions; but that deep sutures to safeguard against hemorrhage may carry infection deeply into the tissues, where it will become of serious consequence, as the investigations we have just heard suggest as possible, must be seriously considered.

It is, undoubtedly, the prevailing opinion at the present day that the lung abscesses following tonsillectomy are of aspiratory origin. The communication we have just heard gives us good grounds for believing that embolism from original thrombosed vessels is an important factor and accounts for a certain proportion of cases. I doubt if anyone who considers the question in a wholly unbiased way would deny that some cases are aspiratory and others embolic. Unless our whole pathologic belief is wrong, either is a reasonable possibility, but it would be hazardous to venture an opinion as to the relative frequency of aspiration *versus* embolism. The practical conclusion, so far as I can see, is that those engaged is this sort of surgery should strive to devise technic that will reduce the danger of both of these possible accidents.

Dr. Fetterolf (closing): The writers have enjoyed greatly the discussion of their paper and want to thank Dr. Opie, Dr. Jackson and Dr. Stengel for coming here. As was said in the early part of our paper, we approached this problem with no preconceived ideas. I had one case of posttonsillectomy pulmonary abscess which died, and it was a distressing experience. Now that the frequency of this complication is becoming better known, Dr. Fox and I wanted to dig into it and find out whether anything further could be devised to prevent it. When we did these experiments and looked over the specimens which have been shown you on the screen, we became a little stirred over the possibilities. Undoubtedly there is a certain proportion of cases which are of inspiratory origin, but we do feel that we must give more consideration to the embolic side. Almost everybody has been emphasizing the possibilities of inhalation, but very few refer to embolism. We believe that more emphasis should be laid on the embolic potentialities, with the idea of making the tonsil operation more safe and thus lessening the mortality. Stress has been laid upon methods of preventing blood, mucus, etc., from entering the trachea. We believe that this is as it should be, but we further believe that greater attention should be paid to the operation area. We have drawn a few practical deductions, and we really do not care how many believe in the inspiratory or how many believe in the embolic origin. The important thing is by every means at our command to diminish the frequency of this complication.

THE GALL-BLADDER: ITS PAST, PRESENT AND FUTURE[1]

The Mütter Lecture

By J. E. SWEET, A.M., M.D., Sc.D.

The method of study of a problem in medicine is like the method employed in the construction of a modern mechanical appliance. Different materials enter into the mechanical structure, coming from widely separated areas of the earth, passing through the most diverse processes, to be finally assembled into a form familiar to us in every-day life. Such is the ordinary telephone receiver, into which has been wrought rubber from the tropics, platinum from the Urals, cotton of the southland, Colorado copper and mica from India. These raw materials, skilfully smelted, spun and baked, are finally assembled into a functioning entity—none may be left out.

And so in the working out of the problems of medicine: Many are engaged in the collection of the raw materials—zoölogists, embryologists, comparative anatomists, physiologists, pathologists, practitioners of medicine and surgery; many are skilfully smelting, spinning and baking. Had I any serious criticism it would be that too few are engaged in assembling, in putting together what we have, to see not only if the pieces fit, but to learn what sort of things are still lacking, and possibly what their nature and where they must be sought.

It was with this thought in mind that I chose the problem of the gall-bladder, for it seems that there is a good deal of raw material

[1] Read December 7, 1923.

in the way of knowledge concerning the gall-bladder, but nowhere have I found it assembled. Let us then collect this knowledge and see what sort of an apparatus we might construct of the materials at hand. Even if we cannot decide just what sort of work it will do, perhaps we can gather an idea of what sort of work it will not do.

A further thought determining my choice of a subject which might hope to take its place in the notable list of the Mütter Lectures was that a subject concerning which all is not yet known may prove of greater interest to the audience, by its offering of food for thought than might a finished subject.

THE GALL-BLADDER, ITS PAST, ITS PRESENT, ITS FUTURE. The past of any human organ is written in the history of its development: We may study the lower forms and expect to find the details of their anatomy represented in the stages of the development of the highest form; we may expect to encounter in the human, in the form of anomalies, persisting stages or atavistic reminders of the lower orders; or we may study the human embryology and read out of it the details of the anatomy of the lower forms.

The present of the gall-bladder is the actual structure as we see it in gross and under the microscope.

THE FUTURE OF THE GALL-BLADDER. Who shall prophesy the future? Yet we are taught that the future of the individual is determined by the kind of a life which that individual leads. So if we can obtain an inkling of the sort of a life the gall-bladder leads, of what it does in this world, whether its work be good or evil, perhaps then we may venture a prophecy as to whether the gall-bladder is destined to share the heavenly pedestal upon which the pancreas now rests supreme—a pedestal with *nolo me tangere* written large upon it, rests safe from the surgeon's knife if not from his prying finger—or whether the gall-bladder is doomed to fall into the hell of pathology, there, pickled in alcohol, to form a scorned triumvirate with the tonsils and the appendix!

At a very early stage of the embryo, when the gastrointestinal tract is composed of the foregut, the midgut connecting with the

yolk-sac and the hindgut, a fold of the wall of the foregut appear just above the junction between the foregut and midgut. This fold, formed just as one might pinch up a fold of one's sleeve between the thumb and finger, represents the beginning of the liver and the gall-bladder. It is spoken of in the literature as a diverticulum, or as a groove, or a furrow. The discussion which has appeared in the literature as to whether the liver develops from one or from two anlagen (a German term still in common use by embryologists, in spite of the late unpleasantness, to designate the first aggregation of cells which will form any distinct part or organ of the embryo) seems to have been settled in this sense that one primary groove appears out of which two structures develop. From the upper, the cranial end, the liver arises, soon growing out as numerous buds, which grow around the dorsal vein, thus embedding the future hepatic vein within the liver substance; from the lower, the caudal end, develops the gall-bladder.

Hammar has given us reconstruction models of the development of the liver and the gall-bladder of the chick. It is to be noted that in the chick both buds retain their continuity with the intestine, thus forming separate channels of entry into the intestine for the bile from the two lobes of the liver, a condition which persists and characterizes the avian type of biliary apparatus.

In Hammar's[1] models of the developing liver of the rabbit the liver anlage seems to have pushed away from the bowel, so that it remains attached to the bowel only by a common duct which has developed from the caudal bud or gall-bladder anlage.

Such seems to be the consensus of opinion of those who have worked with the subject—that the liver and the gall-bladder arise as separate structures, closely related, coming from the same fold of the foregut, but distinct entities, nevertheless (Hammar,[1] Brachet,[2] Brouha,[3] Broman,[4] Rietz[5]).

While this point of the embryologic independence of the gall-bladder is emphasized by those who have worked on the subject, there are several facts which will later be demonstrated—the similarity of structure between certain appendages of the bile-duct and the gall-bladder and the possible enlargement of these struc-

tures after removal of the gall-bladder—which incline me to look upon the gall-bladder and the duct system of the liver as a unit, which may in the main arise as an independent embryologic unit. From this point of view the gall-bladder would be an appendage of the duct system, but not differing, except in form and location, from the ducts themselves. Whichever way we may care to consider it, this remains true, that the gall-bladder develops from that group of ancestral cells at the lower end of the foregut which gives birth to such important structures from the standpoint of absorption and of secretory function as are the stomach and the duodenum, the liver and the pancreas.

This, of course, does not mean that an organ of simple function could not be built out of such raw material. Yet it does not seem likely to me that Nature would take from such potential material for the construction of a simple sac or reservoir. Nature, the great conservationist, would not take from a gold mine to make a tin can. That she can, on the contrary, transmute an old tin can into purest gold is seen from what she made from Adam's rib!

From this early start the gall-bladder and its duct develop in much the same way in all species. There is, indeed, some evidence that those species which have no gall-bladder do show these early stages of its development (Rietz[5]). At first the bud growing from the intestinal wall is hollow; then the cells lining this little cavity seem to develop so rapidly that the structure becomes solid, to hollow out again later. It is at this stage that certain of the recorded anomalies of the gall-bladder arise; the cystic duct becomes hollow, but the gall-bladder fails in its duty, so that a cystic duct appears, but no gall-bladder. The walls of the gall-bladder stick together through the middle so that a gall-bladder with a septum results, or even a double gall-bladder. The gall-bladder hollows out, but the duct remains a solid strand. It is a noteworthy fact, however, that very few such anomalies have been recorded. Congenital absence of the gall-bladder in those species in which a gall-bladder normally occurs is very rare.

Such is the general manner of development of the gall-bladder throughout the kingdom of the vertebrates. Differences in detail

appear which relate to the final location of the gall-bladder, and more particularly to the manner in which the gall-bladder connects with the duct system of the liver.

An interesting location of the gall-bladder is found in the true serpents, where the ducts are very long and the gall-bladder has been removed a long ways from the liver. Owen[6] explains this by the fact that in the true serpents the prey is usually of large bulk; this enormous distention of the stomach would exert pressure upon the gall-bladder were it in the usual location, and so Nature has removed the gall-bladder to a site beyond the stomach. Another curious condition is found in some reptiles, as in python, where the cystic duct, single at its commencement, divides into numerous branches which penetrate the pancreas and reunite with each other and the hepatic before terminating in the duodenum.

Such differences as this most pronounced one between the location of the gall-bladder in serpents and the location of the human gall-bladder are not basic differences—they are determined by the differences in adult form and anatomic arrangement rather than by differences in the embryology. Therefore, we do not find such anatomic peculiarities appearing as anomalies in the human being.

The manner in which the duct system of the liver and the gall-bladder connect is a matter, however, of embryology. We have seen how the liver anlage of birds remains attached to the intestine by its own duct, while in the rabbit the duct system of the liver and the gall-bladder unite to form a common channel into the intestine. Slight variations in these arrangements occur in various species, so that four types of duct systems have been described. In nearly all birds the arrangement is such that the cystic duct comes exclusively from the right lobe, the left liver lobe draining by an entirely separate duct directly into the intestine. The two systems are, however, connected by radicals within the liver.

That atavistic reminders of earlier types are not at all uncommon in the human being is shown in Flint's study[7] of the cystic artery and the duct arrangement in 200 consecutive autopsies. Variations occur in the length of the cystic duct and in its mode of

union with the hepatic duct, conditions reminding one of the avian type are found. Certain points of great practical importance to the surgeon are emphasized by Flint's work, the rather common occurrence of accessory ducts from the right lobe, either crossing behind the cystic duct to the hepatic duct, or to the common duct, or to the cystic duct—conditions reminding one of the anatomy of the bile ducts of the dog. It is probable that the cutting of these accessory ducts during the cholecystectomy is responsible for the cases in which bile leakage occurs rather than the slipping of the ligature of the cystic duct.

Such is the past of the gall-bladder and such is the rôle of that past in the explanation of the anomalies encountered. From the source I have pointed out, that segment of the embryonic foregut so rich in potentialities, by the complex processes of fetal growth, there has arisen as an independent unit the gall-bladder, or perhaps the gall-bladder and the duct system of the liver.

What is the structure of the gall-bladder; what is its present? The human gall-bladder is pear-shaped, about the size of a hen's egg, lying in its groove on the under side of the right lobe of the liver near the anterior edge; hanging by its stem, the cystic duct, but also fastened in a varying fashion to the liver surface. It is usually fastened over nearly one-half its surface to the liver and may be entirely surrounded by liver tissue except the tip, in some animals regularly so. It may be entirely free, hanging by a true mesentery.

Two points stand out in this gross arrangement which bear upon the possible function of the gall-bladder. With the exception of those rare cases in which the gall-bladder hangs entirely free in the abdominal cavity, the gall-bladder is so attached by one-third to one-half of its surface to the liver that it could never be entirely emptied by the activity of its muscular wall, assuming for the moment that such is the function of the muscular coat of the organ. When the gall-bladder lies entirely buried within the liver tissue it could hardly be emptied at all by such activity of the muscular coat. The second point to note is that the relatively fixed part of the gall-bladder is the neck and the cystic duct,

which is attached through the fusion of the ligaments supporting the stomach, the duodenum, and the colon to the tendinous portion of the diaphragm. Therefore, any force which tends to displace the anterior edge of the liver downward, such as a tight corset or visceroptosis, will tend to increase the angulation of the gall-bladder on the cystic duct, a factor which has been emphasized in explaining the tendency to gall stones, which seems to be associated with corsets and visceroptosis. It is usually stated in this connection that such displacement prevents the emptying of the gall-bladder, but I would emphasize that such displacement would be equally efficient in preventing the filling of the gall-bladder.

Why is the gall-bladder situated where it is, beneath the free edge of the liver, where it is subjected to pressure with every inspiration? I think not necessarily in order to empty the organ, for, having seen the lymph flow along a cannula placed in a lymphatic of the gall-bladder, the column of lymph being pushed onward by every inspiration, I may point out the possibility that the gall-bladder is made subject to the rhythmic pressure of inspiration to aid the flow of lymph and of the venous blood. It is too often forgotten that the main driving force of the lymph from periphery to center and an important adjunct to the return of venous blood is the activity of the skeletal muscles; so in the intra-abdominal organs some force other than *vis-a-tergo* is needed for driving the lymph along, such as the peristalsis of the intestine. Lymph ceases to flow freely from the thoracic duct when respiratory movements are stopped; and I suggest that this is as plausible a reason as any other for placing the gall-bladder where it is placed.

The angulation of the cystic duct resulting in an S-shaped curve or kink has not been given due consideration in respect to its bearing on the problem of the possible function of the gall-bladder. The manner of origin of this curve or kink has been explained as follows: The cystic artery, growing on to the gall-bladder from the hepatic artery, attaches to the gall-bladder at an early stage and offers a certain degree of fixation of the gall-bladder; as growth continues and the cystic duct lengthens, it cannot straighten out because of this fixation of the gall-bladder by the cystic artery,

and, therefore, it becomes thrown into curves. Whatever be the explanation of its origin, the mechanical effect of such a kink must be to constitute a sort of valve—pressure on the filled gall-bladder kinking and shutting off the cystic duct, preventing the emptying of the gall-bladder, but being equally efficient in preventing the further filling of the gall-bladder.

This gross valve action seems to be increased by the spiral fold of the mucous membrane lining the duct and the neck of the gall-bladder of the human being, a curious structure which has long been known, but to which little attention has been paid in the literature and no attention at all in the discussions of the possible function of the gall-bladder. This spiral fold was first described and pictured by Lorenz Heister, called the "Father of German Surgery," who lived from 1683 to 1758, and it is still known as "Heister's valve." From Heister's own words, however, it would seem that such a structure in the cystic duct had long been under discussion, for Heister[8] mentions that Glisson, the English anatomist, who died several years before Heister was born (1559-1677), had never found such a valve.

"Glissonius (*Tract. de Heapate*, cap. 14) quidem ait, Anatomicos quosdam in ductu cystico valvulas effinxisse, se autem credere fibrosum annulum cystidis iis invenisse, se enim nunquam valvulam ibi invenisse. Ita & Bianchus (*Hist. hepat.*, cap. 14, p. 2) inquit, valvulas cystis felleæ collum & cervicem occupantes, imiginarias esse. Verum an non hæ indicatæ partes, non semel, sed ab aliquo tempore in plerisque cadaveribus repertæ, sint dicendæ valvulæ, aliis dijudicandum reliquo. Ita & Quem usum hic præstare queant, doctoribus indigandum commendo."

The old anatomical method of preserving hollow viscera for permanent specimens—blowing them up with air and drying them—is possibly the best method of demonstrating the valve of Heister. The abandonment of this crude method with the advance of medicine may explain why the structure has escaped the prying eye of modern generations. Hendrickson[9] seems to be the only one of recent years who has studied this valve-like device. It is a fold of the mucous membrane arranged in spiral form, with the

larger folds in the neck of the gall-bladder, the folds growing less marked down the duct, exactly like the thread of a wood-screw, with the head of the screw in the gall-bladder, the point in the cystic duct. But this device is more than a fold of a membrane; the folds contain muscle fibers, arranged longitudinally and transversely, seemingly placed to manipulate the valve leaflets.

Heister[8] decided to leave the discussion of the purpose of this structure to someone else, and apparently each generation since has followed his lead. The only suggestion I have encountered is the one offered by Keith,[10] who in discussing the relation between enteroptosis and gall stones, says that in enteroptosis the gall-bladder is so displaced that the fundus and the common duct tend to lie parallel instead of at an angle of 35 degrees to 45 degrees, a strain at the same time is thrown on the cystic duct, "which even in normal conditions requires a spiral valve to keep it open." With this thought I cannot agree, as the valve of Heister is too delicate for such purpose. The only explanation of these two devices—the cystic kink and the valve of Heister—which appeals to my mechanical sense, is that they are designed, and efficiently designed, to hinder the emptying of the gall-bladder by external pressure.

I presume the time will yet come in these days of such rapid surgical advance when some youthful enthusiast will put on the market an operation for the relief of Heisterian spasm!

The gall-bladder is ordinarily described as being made up of four layers: First, an external covering of peritoneum over that portion of the organ which is not attached to the liver; second, a layer of connective tissue in which, but near, the mucous lining; third, a layer of delicate muscle fibers running in an interwoven layer of circular, longitudinal and diagonal strands. The lining (fourth) is a single layer of tall columnar epithelium. Throughout the connective tissue which holds the layers together run blood-vessels, lymphatics and nerves.

The peritoneal covering and the connective tissue of the gall-bladder merit no special attention. The muscle coat, however, deserves closer study, because of its relation to the possible func-

tion of the organ and because so much has been said of it in recent years. It is not a powerful affair, but a delicate mechanism. All those who have especially studied it seem united in the opinion that it is a muscularis mucosæ—that is, a layer of muscle fibers more intimately related to the mucous membrane than to deeper-lying structures, the function of which is to throw the mucous membrane into folds and to vary the size and shape of these folds to move the lining membrane around over the content of the viscus (Hendrickson,[9] Sudler,[11] Shikinami,[12] Berg[13]). Hendrickson shows a cross-section of the wall of the gall-bladder showing the interweaving course of the muscle fibers. Berg shows pictures of gall-bladders prepared by maceration, which show very clearly how the characteristic reticulated appearance of the interior of the normal gall-bladder is determined by the course of the muscle bundles lying close beneath the single layer of columnar lining cells.

These folds are pictured by Boyd,[14] who rises to poetic heights in his description of them: "In text-books of anatomy one reads the statement that the mucous membrane of the normal gall-bladder is thrown into folds. But this conveys little to the mind until the gall-bladder is viewed direct under the dissecting binocular microscope. When the fresh gall-bladder, immersed in water, is observed by reflected daylight, or, still better, by the brilliant light of an electric arc, the picture is a remarkable and beautiful one. As if one were gazing into the depths of a marine pool at sea weeds and sea anemones, tall, graceful folds and membranes, gossamer-like in their delicacy, can be seen floating in the ambient fluid. The entire inner surface is divided by these membranes into a series of polygonal spaces, and each of these spaces resembles a little courtyard surrounded by high, though delicate, walls." And I would add thereto, and emphasize, that the walls of each little courtyard can be moved about by the muscle bundles which determine them.

What of the rhythmic contractions of the musculature of which much has been heard in recent years? (Bainbridge and Dale[15]). So far as I am able to determine, they are only the rhythmic movements which are common to smooth muscle and are of no impor-

tance as an agent in emptying the organ. This type of movement, particularly of a muscularis mucosæ, might well bring fresh surfaces of mucosa in contact with the content and move the content about so that the fluid in the center would be brought into contact with the mucosa; such movement would also help in the driving onward of the lymph in the walls of the organ.

Within the tissues of the gall-bladder wall course bloodvessels nerves and lymphatics. The arterial supply is from the hepatic artery through the cystic artery; the veins empty into the portal vein. According to Beale,[16] the arrangement of the vessels of the gall-bladder is very peculiar: "Each branch of artery is accompanied by two branches of the vein, and this arrangement exists even in the case of very small divisions; the small branches of the arteries anastomose very freely, in some cases forming five- or six-sided spaces, so that an arterial network is formed. This is wonderfully developed on the external surface of the gall-bladder. Each artery composing this network is accompanied on either side by a branch of vein. . . . The wonderful arrangement of the venæ comites is not easy to account for, but it is very remarkable, and it is found to the extent here demonstrated as far as I am aware in this organ only." A very rich blood supply also passes to the bile-duct throughout the liver. Sudler[11] emphasizes the "exceptionally rich capillary network" beneath the folds of the mucous membrane of the gall-bladder. Luschka[17] mentions the rich blood supply of the edges of the folds.

In the crypts formed by the folds, just under the mucous membrane, solitary lymph follicles are found, with an elaborate plexus of intrinsic lymph channels in the submucous tissue. This work of Sudler[11] emphasizes very strongly the extent of the development of the lymphatic apparatus of the gall-bladder; a similar close relation exists between the mucosa of the bile-ducts and the blood and lymph supplies. This intimate relation between the biliary system and the lymphatic system assumes a real importance to surgery and surgical pathology when we stop to think that every localized infectious process is a lymphangitis.

The activity of the lymphatics has been demonstrated in our

laboratory by introducing into the gall-bladder a hypertonic solution of potassium sulphocyanide, then collecting lymph from fine tubes inserted into the lymph vessels of the gall-bladder and testing the lymph obtained with ferric chloride. In fact, working as rapidly as possible, the first specimen procured gives a positive Prussian-blue reaction (Harer, Hargis and Van Meter[18]). This lymph from the wall of the gall-bladder also gives positive chemical reactions for bile pigments and bile salts.

The gall-bladder, according to Freese,[19] receives both motor (constrictor) and inhibitory (dilator) nerve fibers from the splanchnic nerve. Sympathetic fibers doubtless follow the artery.

There still remains the fourth component layer of the gall-bladder to be considered; and if we have any reason to suspect from what we have seen thus far that the gall-bladder is something more than a mere reservoir or a pressure-regulating safety device, then this layer deserves especial notice, for it would be by the activity of the lining cells that any function other than a mechanical one would be carried out.

The mucosa of the gall-bladder is composed of a single layer of rather high columnar cells, by some described as containing granules in their protoplasm toward the free edge of the cell. These cells are comparable to the cells lining the intestinal tract, and just as in the intestine the total surface area is enormously increased by the folding of the membrane. In the intestine this folding is regular and the folds are known as villi; in the gall-bladder the folds are less regular, but more delicate. A few glands, it is said are found in the gall-bladder, and these mostly near the neck. The glands described as Luschka's glands are simply outpouchings of the mucosa through defects in the muscular coat. In fact, in view of certain structure presently to be described, it is doubtful whether any true glands are to be found in the gall-bladder wall.

This same type of high columnar epithelium, marked by stippling or striæ of the free edges, is found in another place in the body. May I ask your indulgence to leave the gall-bladder for a moment and wander up the bile-ducts, for the bile-ducts are lined with this same type of cell. Another cell is pictured as occurring with them

in the bile-ducts, a larger cell without the striation. Whether this cell really represents a different cell or only a different stage of activity of the same cell is not clear.

If we go back to the literature of a hundred years ago, before the day of the microtome and the aniline dye, to those days when men were trained in the use of the most perfect optical instrument yet devised—the human eye—an instrument which possesses another striking peculiarity beyond its perfection, that it is sometimes associated with the brain, we will find two structures described which later anatomists have either never seen, or seeing have lightly passed over. The days I speak of were the days of anatomy when injection and corrosion preparations were in style, a technic little used today, but possessing certain advantages.

The inside of the larger bile-ducts is found to be provided with minute depressions, in the human arranged in parallel rows. These depressions, known to Haller, and called "scrobes depressæ" by him, open into two structures; the one is the blind-ending ducts discovered by Ferrein and called "vasa aberrantia." These seem to be the remains of previously functioning ducts, the liver tissue which they drain having been lost through pressure atrophy; they are most numerous along the portal fissure, and are found where pressure atrophy has apparently occurred. The vasa aberrantia have not been entirely lost to sight, but the other structures into which the scrobes depressæ open have disappeared from modern text-books, or perhaps are lightly passed over as mucous glands. I refer to the little sac-like appendages discovered by Theile, called by him without any reason I am able to find in his description "Schleimdrüsen," perhaps best called "parietal sacculi," which are found all along the bile-ducts within and without the liver, differing somewhat in their form and number according to the individual and to the species, but not found along the common duct below the opening of the duct of Wirsung, a fact of interest in those animals in which the duct of Wirsung opens very high up into the common duct.

A cross-section of the empty, collapsed duct shows why these

structures do not appear in preparations made by modern methods; it shows only a folding of the mucosa with cross-sections of structures which might be glands or which might be but the edges of the neighboring folds of the mucous membrane. The question of whether these structures can be brought out by the influence of chronic irritation is raised after studying the localization of liver parasites. Structures very suggestive of the possible accentuation of the parietal sacculi by the presence of the parasites can readily be found.

The most extensive description of these parietal sacculi is found in Beale's work, in which is also found the refutation of the assumption that they are mucous glands. Beale finds that the bile of the rabbit, in which animal the sacculi are almost absent, contains as much mucus as does the bile of the pig, in which animal they are exceedingly numerous and are arranged entirely around the ducts. These little pouches lie within the fibromuscular wall of the duct. They have an abundant blood supply, and are in such close relationship with the lymphatics that the lymph vessels are commonly injected in the course of demonstrating the saccules.

As to the function of these structures, Beale[16] remarks: "In fact, I think that we may almost regard them as supplementary little gall-bladders appended to the ducts." With this thought in mind it is interesting to compare the bile-ducts of the horse with the parietal sacculi, an animal which has no gall-bladder, with the same structures from the pig and cat, which animals have gall-bladders.

And now, if the future of an organ of the body is to be determined on the same principle as determines the future of the collection of all the organs of the body—the individual—that is, by the kind of a life the individual leads—what does this organ, the gall-bladder, do? This little sac, growing from an ancestral cell group filled with potential possibilities, growing independently of the liver in an embryologic sense, developing a blood supply characterized by its richness and a peculiar arrangement of the veins, possessing an unusual lymphatic drainage, provided with a double valve device at its outlet manifestly designed to prevent outflow,

possessing a lining membrane composed of cells comparable only to the cells of the intestine whose powers of secretion and absorption have never come into question—assuredly we must begin to suspect that a structure so complex is made for something other than storage or than a pressure-regulating device!

The common concept of gall-bladder function is not new; it is the same as was expressed by Heister just two hundred years ago, and there is no evidence in his writings that he considered his explanation anything new. Yet in Heister's definition, as in many which appear throughout literature since his time, there is suggestion of something deeper, something beyond mere mechanics, *ulterius eam perficere*. Thus Owen[6] adds to his mechanical explanation of the curious location of the gall-bladder in serpents: "The gall-bladder is not, however, a simple reservoir; its vascular and secreting inner surface can operate upon the bile by both subtraction and addition."

So much has been written, so many suggestions discussed—and cussed—that perhaps the best method would be to wipe the slate clean and start anew. Even though the process involves something which seems to cause acute spinal shudders to so many—the introduction of a new theory! These I will comfort ere I start by saying that we may not find the theory so entirely new after all.

We have alongside the upper intestine the pancreas, which elaborates and pours into the intestine an excretion which has to do with the breaking up and the absorption of the proteins and the carbohydrates. The products of this digestion are taken up through the intestinal wall into the blood stream. Closely associated with the pancreas is another organ, the islands of Langerhans, the secretion of which is added to those products of digestion within the blood stream and before this blood in the portal vein has reached the liver. In a similar relation to the intestine stands the liver, pouring into the lumen of the intestine an excretion which has to do with the absorption of fats. These products of fat digestion pass through the intestinal wall into the lymphatics. Closely associated with the liver is another organ, the gall-bladder,

which may in analogy with the activity of the islands of Langerhans add something to the lymph which is carrying the products of fat digestion before the lymph passes into the general circulation. The geographic analogy is complete; we have seen that the gall-bladder possesses an abundant lymph supply and that the absorption through these lymphatics is very rapid. Is there any evidence that our analogy holds good, that the gall-bladder has anything to do with the metabolism of the fats?

Many writers have sought some suggestion of the function of the gall-bladder in a comparison of those species which possess gall-bladders with the species which have none, but they have abandoned the attempt on finding closely related individuals in both groups; for instance, the two-toed sloth has a gall-bladder, the three-toed sloth has none. But in a broader way there is a difference. The true meat-eaters possess gall-bladders, while the varieties lacking this organ are, in the main, herbivorous. The main element present in a meat diet and not found in a vegetable diet is animal fat, and certainly the fat element of the diet is almost entirely lacking in the grass-eaters.

There is, further, I believe, good clinical reason for assuming a relationship between the gall-bladder and fat metabolism. That famous slogan of Philadelphia's master of the gall-bladder and of epigram, "Fair, fat and forty—gall stones," is a fact universally admitted. That, in general, gall stones occur in people who are overweight; that is, who are possessed of some abnormality of fat metabolism. Further, many patients put on weight after cholecystectomy; and patients without a gall-bladder are often unable to handle a fat diet and are commonly cautioned against eating fat. According to Mikaya, gall stones occur in a much lower proportion of Japanese women than women of the Occident. We are accustomed to about five women to one man. In Japan, the ratio is three to two. This may or may not be due to the fact that Japanese women do not wear corsets; of more immediate importance is the fact that the gall stones encountered in the Japanese who are vegetarians are pigment stones, not cholesterol stones.

And now for the comfort of those who dread a new theory. In 1846 Rudolph Virchow[22 a] described the gradual filling of the cylinder cells of the gall-bladder mucosa with fine fat granules. In a later article[22 b] he speaks of an intermediate metabolism of fats in the wall of the gall-bladder; he compares the process with the absorption of fats by the cells of the intestine, a comparison supported by the striking similarity between the mucosa of the intestine and the mucosa of the gall-bladder.

"As regards the filling (of the cells of the mucosa of the gall-bladder) with fat, it is comparable to a hair with the resorption of chyle by the intestine. At first very finely granular fat appears; later large, glistening drops. At first the fat appears in the upper layer of the cell, close beneath the homogenous edge, while the deeper portions are still free. Then it gradually advances deeper, until the entire cell, with the exception of the nucleus, is filled and stretched. It is at this time, when the fat droplets lie in rows so that they form parallel strings of pearls from cell cover to cell peak, that an appearance is imparted to the cell like that of the fatty degeneration of a muscle bundle. That fat then disappears from the outer portions of the cell, and one finally sees only the base still saturated with fat droplets while the surface and the center are entirely free" (*loc. cit.*, p. 576). Virchow believes that one must conclude from this observation that the gall-bladder possesses a further function than that of a mere reservoir, and that this function is that of taking fat from the bile and restoring it to the circulation, together with other things.

In 1908, Aschoff,[23] in a paper "in regard to the formation of cholesterol by the gall-bladder," was unable to confirm Naunyn's belief (no reference given by Aschoff) that the epithelium of the gall-bladder produces cholesterol. He did show by experiment that the epithelial cells of the gall-bladder can take up neutral fat from the content of the gall-bladder, and that this fat is found in the lymphatic, he states that cholesterol esters can be taken up in the same way. He thinks that this phenomenon is of importance because the fats are solvents of the cholesterol, and the removal of the fat from the gall-bladder content would favor a precipitation of the cholesterol.

In 1914, Policard[24] concluded that a fat absorption takes place in the gall-bladder in a manner entirely comparable to the process in the intestinal epithelium. Successive stages can be followed, and there is an alternation of function not between individual cells, but between different areas of the epithelium; this alternation of function is dependent on changes in the circulatory apparatus.

In 1923, Boyd,[14] using all the resources of modern science, concludes that the fat-like substance found in the cells of the gall-bladder mucosa is an ester of cholesterol, and he advances the suggestion that the function of the gall-bladder is one of absorption, and that possibly one of the chief substances absorbed is the cholesterol of the bile.

There are in the literature many other references to the relation of the gall-bladder to cholesterol metabolism; it would lie beyond the scope of this paper to attempt a complete analysis of this literature, nor would the gain be great. We were, however, prompted to try the following experiment: To feed a normal dog a known fat meal and to determine the cholesterol content of the blood at definite intervals thereafter. This work was made possible by the collaboration of Dr. George W. Wagoner, and we are greatly indebted to Dr. W. G. Karr, of the department of chemistry of the laboratory of the Philadelphia General Hospital, for his constant help in the envolved chemistry. After an interval the gall-bladder was removed, and then at certain times the procedure was repeated, giving the same known fat meal at the same hour before each experiment. The results appear in the accompanying Curve No. 1, showing the results obtained in one animal, and in Curve No. 2 a composite curve of four normals and three cholecystectomies.

Two things are apparent from these curves: The total blood cholesterol rises very noticeably immediately after the removal of the gall-bladder; it rises to almost double the normal and then falls to the normal level forty days after operation, and is considerably below normal at seventy-four days. We have one observation at three hundred days after operation, when the curve approaches the normal. The second noticeable fact is that the

peak of the rise following fat ingestion occurs normally at the sixth hour; whereas, after the removal of the gall-bladder the peak is reached at the ninth hour.

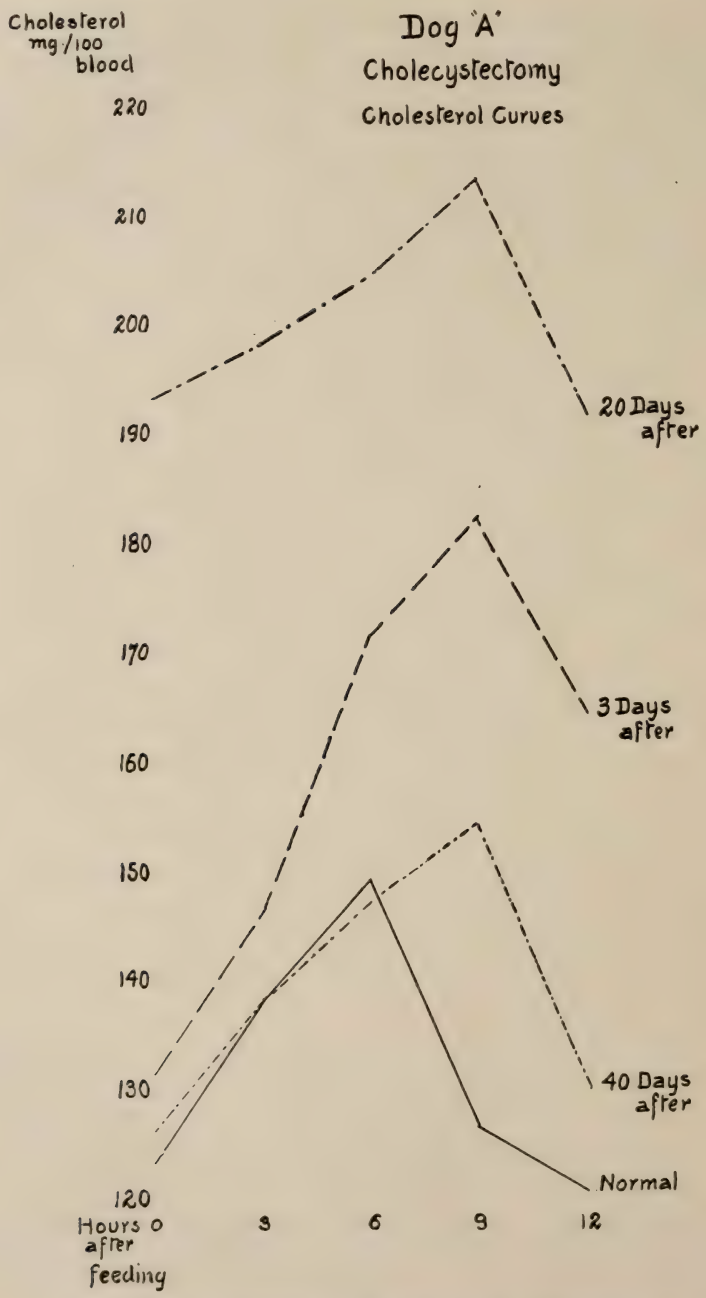
There seems to be always a certain balance preserved in the body between the two blood lipoids, cholesterol and lecithin; we, therefore, in the beginning of our work, included a determination of lecithin. The results in one animal appear in Curve No. 3. The curves show that the lecithin content of the blood is not much disturbed, the difference which appears in this curve being more apparent than real. Yet the same three-hour delay in attaining the height of the curve is shown by the lecithin.

An enthusiastic appraisal of these curves might lead to many suggestions; a modest evaluation would be that they show very clearly and conclusively that the removal of the gall-bladder does exert a considerable effect upon the metabolism of cholesterol.

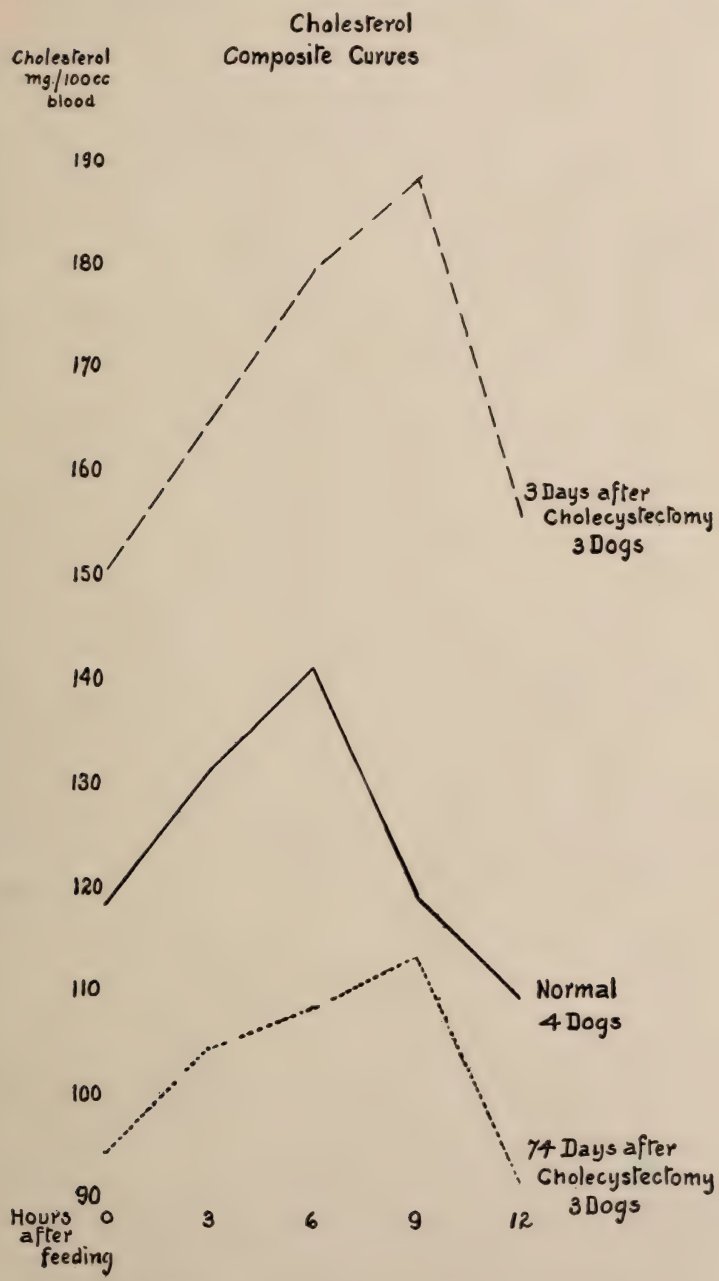
We know that cholesterol occurs in the blood in two forms—as free cholesterol and as an ester or combination of cholesterol and some fatty acid. We have seen, from Boyd's work, that the lipoid found in the gall-bladder in pathological conditions is a cholesterol ester. Does the removal of the gall-bladder affect both of the forms in which cholesterol appears alike? Is the increase due to an increase of free cholesterol, or of cholesterol esters, or of both in normal proportions?

To determine this point the cholesterol of the blood from a normal animal was partitioned by Dr. Wagoner, the usual fat meal having been given, the usual time periods being observed. The blood cholesterol in four cholecystectomized dogs was then partitioned under the same conditions. The curves of these results appear in Curve No. 4.

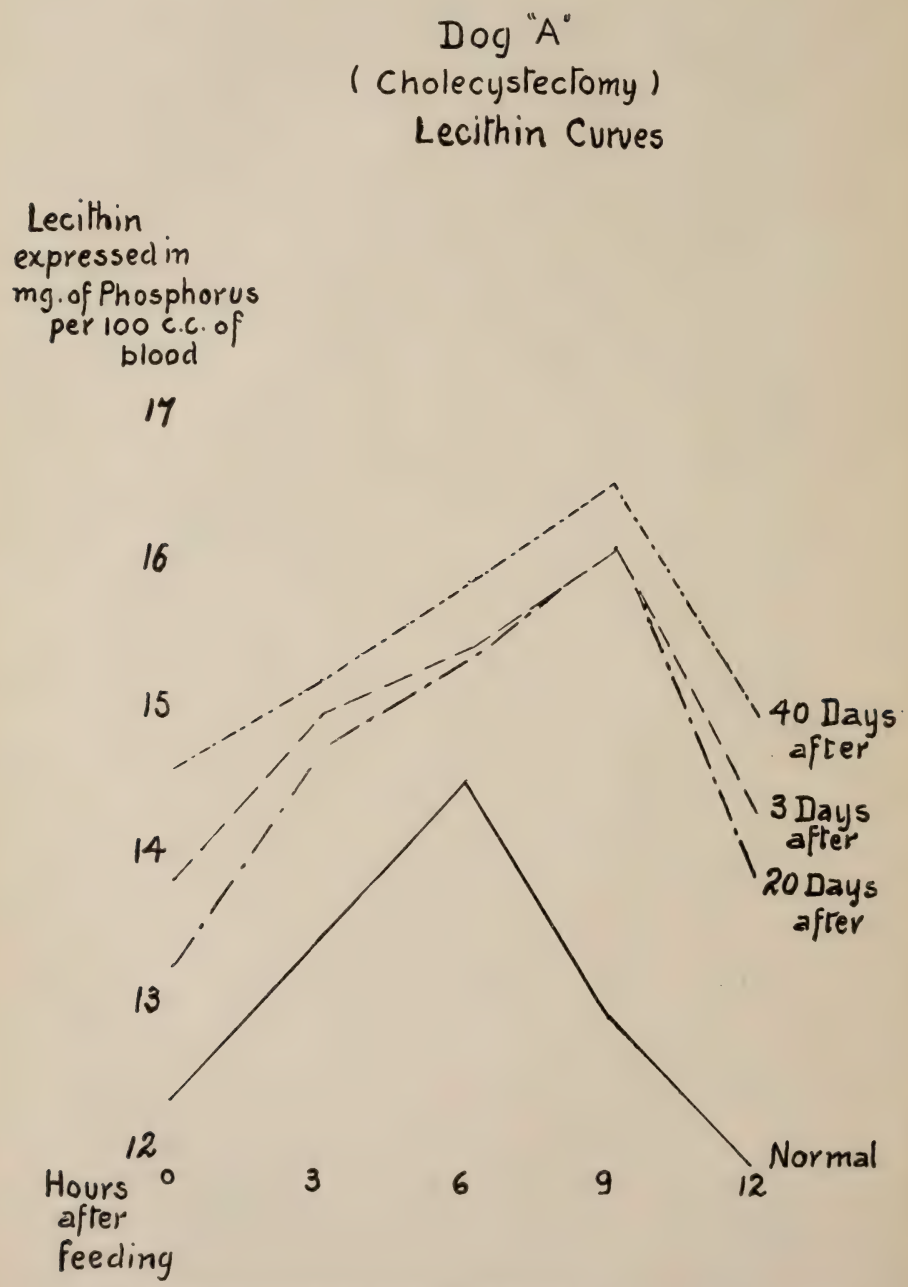
What is this substance cholesterol, and what is its importance to the body? The name "cholesterol" is the newer form of the word "cholesterin," the chemists having adopted the uniform ending *-ol* to designate those bodies which belong to the alcohols. Chemically it is a monatomic alcohol; that is, it contains one alcohol grouping. Physically, it possesses certain of the properties of fat; is soluble, for instance, in fat solvents, hence is classed with



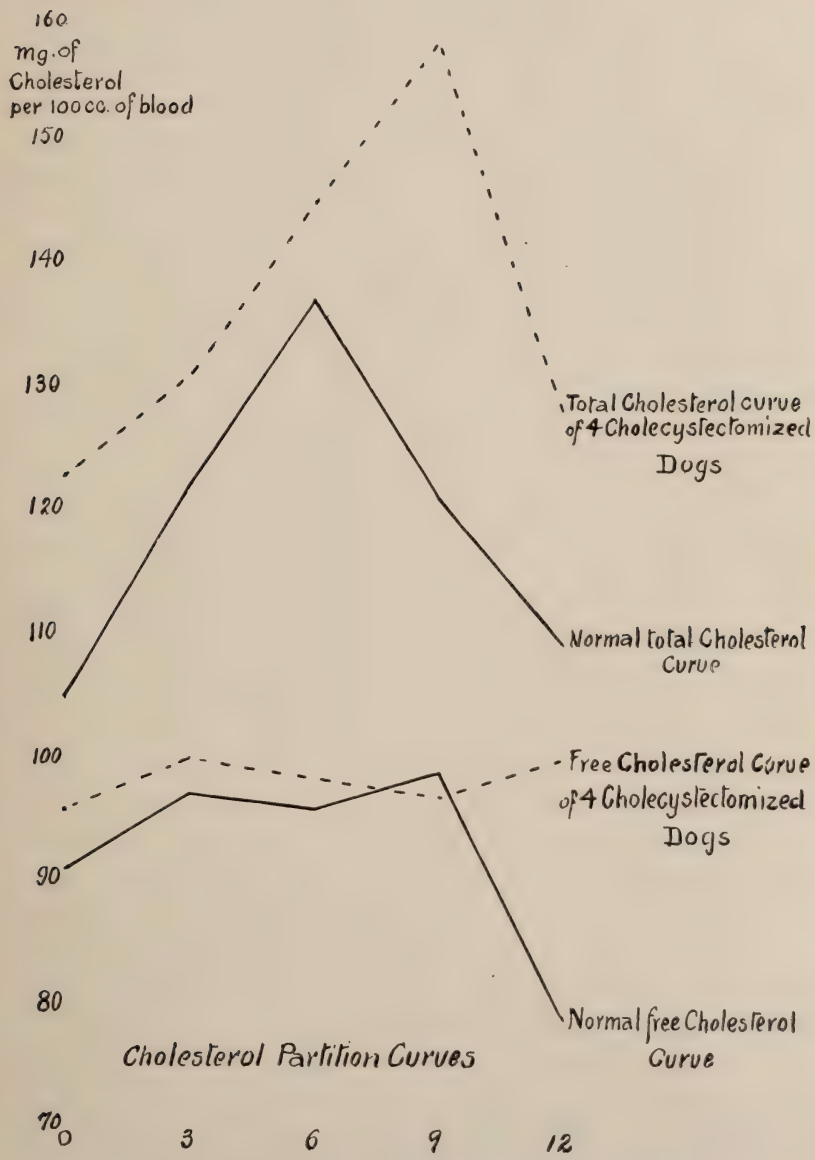
CURVE 1.—The cholesterol of the blood of the normal and the cholecystectomized dog. (Original.)



CURVE 2.—The blood cholesterol: Composite of three normal and four cholecystectomized dogs. (Original.)



CURVE 3.—The lecithin of the blood of the normal and the cholecystectomized dog. (Original.)



CURVE 4.—The cholesterol partition curves. (Original.)

the fat-like substances—the lipoids. Because it is an alcohol, it can form combinations with acids, called esters. Cholesterol is found in all the tissues of the body, the amounts in nervous tissue being noteworthy. The brain contains from 1 per cent to 4 per cent of this alcohol, the higher amounts being found in the white substance. I hesitate to point out this horrible fact, since more than 0.5 per cent is against the Statutes of these United States, and I fear lest the fact being known, a movement shall be immediately started to legislate this illegal percentage out of our systems! As far as we know, cholesterol cannot be built up, synthesized by the body. An isomer phytosterol is widely distributed in the plant world. But very little is known about the physiology of cholesterol; it seems well established that the cholesterol of the bile is carefully conserved and returned to the body.

The tendency of modern physiological chemistry is to feel that these blood lipoids, cholesterol and lecithin, are of importance to the body, entering into the structure of cell membranes; and if such be the case, it behooves the simple-minded surgeon to leave the chemists there, struggling with the problem on the slippery surface of the cell membrane, which membrane they would tell us is, after all, nothing, only the meeting place, the interphase of two liquids!

The greatest stumbling-block to an assumption of an essential function of the gall-bladder has been the fact that no one has ever shown any structure along the biliary system which might take over the supposed function of the gall-bladder when the gall-bladder had been destroyed by disease or removed by surgery. I have dwelt at length on the appendages of the bile-ducts called "parietal sacculi" by Beale[16] because of their similarity to the gall-bladder; they are lined with the same type of epithelium as characterizes the gall-bladder lining, which epithelium bears the same intimate relationship to a rich supply of bloodvessels and lymphatics as we have found exists in the gall-bladder. Analogies so close as led Beale to the suggestion that we must look upon them as minute gall-bladders.

If these structures do have the same function as the gall-bladder,

they should show hypertrophy after cholecystectomy; and the experiment is not difficult of comparing the findings in normal animals with those in animals whose gall-bladders have been removed.

This we have done, being greatly aided in our experiments by Mr. E. M. Landis, of the second-year medical class of the University. The time element in our experiments was based on the curves of cholesterol in the blood after cholecystectomy. In the normal dog we find these parietal sacculæ existing along the biliary ducts, both within and without the liver, as single and compounded structures, varying in numbers and size in different individuals, but in all the normal specimens that we have examined the common characteristic is that these structures are flattened; after the removal of the gall-bladder, at the time when we know from the curves of cholesterol in the blood that the disturbance of cholesterol metabolism has apparently become corrected, the parietal sacculi are found to be changed in their general form. They seem to extend out into the wall. They are longer structures. In many instances the injected ducts of the normal animal look to the unaided eye like perfectly smooth tubes, which show under the microscope these flattened sacculi. But the injected ducts from the animals whose gall-bladders have been removed show to the naked eye as tubes covered with excrescences which give the ducts a sanded appearance.

Our work along this line is just begun, and we do not wish to draw any definite conclusion. Such an investigation must naturally include a study of many different species, both those with and those without gall-bladders.

This demonstration of the existence of normal structures along the bile-ducts with structural potentialities of gall-bladders which I have gathered for you from the literature, together with the suggestion I am able to offer that there is an hypertrophy of these structures after the gall-bladder has been removed—which is, I believe, altogether new—raise many questions in pathology and surgery. The coincidence of gall-bladder disease and bile-duct disease becomes evident—the same pathological process affecting

the gall-bladder extends to these other gall-bladders along the ducts. And when this extension occurs, the surgeon is confronted with this question: While it is perfectly simple to snatch the one large gall-bladder from its home, a home made unhappy by disease it is true, what shall he do with the remaining million gall-bladders which are also made unhappy by the same disease process? Perhaps he had best keep the one big one which he can see and handle for drainage purposes.

What then do I think is the function of the gall-bladder? The manner of origin and development of the duct system of the liver indicates, I believe, that the membrane lining this system may be possessed of powers of absorption; the complex structure of the gall-bladder, the rich blood supply, the unusual lymphatic supply, point to an absorptive function. The position of the gall-bladder, the provision of two valvular structures at its outlet which are mechanically designed to permit inflow and to hinder outflow, lead me to the conclusion that under normal conditions whatever passes into the gall-bladder through the cystic duct never passes out again through the cystic duct. The bile contained in the gall-bladder is eventually absorbed, and in this process of absorption it would seem that the cholesterol esters are broken up, de-esterized. The problem of the importance of this process to the body must wait for its solution until the day when we shall know more concerning the whole problem of the physiology of cholesterol.

It has been to me a real pleasure to collect for this Mütter Lecture the half-forgotten work of old masters, and to revivify it by the work of more recent authors and by our own experiments. The most pleasant duty still remains to me, to thank the committee in charge of this lectureship, and through them the College of Physicians of Philadelphia, for the deeply appreciated honor.

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SPECIAL MEETING OF THE COLLEGE OF PHYSICIANS

December 14, 1923

Introduction by the President

THOMAS R. NEILSON, M.D.

This special meeting has been called for the purpose of having with us, as invited guests, the senior students of the medical schools of Philadelphia. Tonight, then, might be called a student's night at the College of Physicians.

Ladies and Gentlemen, Undergraduates in Medicine, it affords me much pleasure, on behalf of the College, to extend to you a hearty welcome.

We want you to know something about this great institution, the oldest medical society of continuous existence in the United States, dating, as it does, from 1787, and especially we want you to enjoy the opportunity of hearing addresses by eminent gentlemen, who are with us this evening, upon topics of timely interest at the period of your careers which you now have reached.

Here in this hall, the walls of which are adorned with portraits of founders and the former presidents of the College, the temptation is great to stop for a moment to tell you about these men, famed as teachers or as practitioners—some of them teachers of your teachers—whose names are entwined in the history of the College as well as that of American medicine and surgery. The story would, I am sure, be an interesting one. Time, however, forbids me to indulge in the digression. But you are to hear something about this institution.

The first address of the evening will be upon "The Aims and Purposes of the College," by my learned friend, the Vice-President, Dr. Hobart A. Hare, professor of therapeutics, materia medica and diagnosis in the Jefferson Medical College, whom I now have the pleasure of introducing.

The History and Influence of the College of Physicians

By HOBART A. HARE, M.D.

The Committee on Scientific Business has deemed it wise to have a special meeting of this College to which it might invite members of the senior classes of the four medical schools of this city, and the Vice-President has been asked to place before these students certain facts concerning this institution in order that you may be informed of its origin, its accomplishments and its purposes from the time of its formation to the present day.

I would request you to note that, although its Fellows are physicians and surgeons, its correct title is the College of Physicians of Philadelphia, its founders having seen fit to recognize that all surgeons worthy of the name are physicians in fact. It is therefore a College of Physicians and Surgeons *de facto* and the College of Physicians *de jure*.

The College was established and has always been maintained to promote medical knowledge among its Fellows and the profession in general, and with no thought that it would at any time engage in the tuition of medical students. No one is eligible to Fellowship until he has been a graduate at least five years, and the candidate is supposed to have already made contributions to knowledge along medical lines or to give promise of doing so before he is worthy of election. In almost every respect this College corresponds to the Royal College of Physicians, or Surgeons, in London, save that it does not examine and license medical men to practise their profession, and it may be said that no other medical body in this hemisphere occupies quite the same position either as to its age, the purposes of its existence or the care which is taken to limit its Fellowship. Ready at all times to recognize ability, probity and scientific attainment, it nevertheless in these times, when ancient barriers of professional conduct are often pushed aside, insists upon those ideals and precepts that animated its Founders, the pictures of some of whom hang upon these walls.

These pictures are placed not only to note the fact that the originals held high place in the esteem of their colleagues, but for the purpose of letting the men of today gaze upon the types of individuals who have helped to advance American medicine and to impress upon this and future generations that medical men must be controlled by certain ideals as to conduct among themselves and when in contact with the laity.

It is difficult to express the exact meaning of "ideals" in words. "Ideal," when used as an adjective, means something existing in the imagination, visionary or impractical, but when used as a noun it means a mental conception regarded as a standard of perfection, and the latter definition at least marks the goal to which the better type of medical man should aspire, and to this goal this College endeavors to lead its Fellows and indirectly other medical men.

A word as to the history of this College: Its first fully organized meeting was held January 2, 1787, and its Constitution, adopted at that time, states that "The objects of this College are, to advance the science of Medicine, and thereby to lessen Human Misery, by investigating the diseases and remedies which are peculiar to our Country; by observing the effects of different seasons, climates, and situations upon the Human body; by recording the changes that are produced in diseases by the progress of Agriculture, Arts, Population and Manners; by searching for Medicines in our Woods, Waters and the bowels of the Earth; by enlarging our avenues to knowledge from the discoveries and publications of foreign Countries; by appointing stated times for Literary intercourse and communications, and by cultivating order and uniformity in the practice of Physick."

A month later Benjamin Rush, in his first paper before this body, said: "By assuming the name of a College, we shall first be able to introduce order and dignity into the practice of physic, by establishing incentives and rewards for character. Men are generally anxious to preserve the good opinion of those with whom they are obliged to associate. The reception we shall meet with from each other in our meetings will serve to correct or to improve

our conduct. And if we are as chaste as we should be, in the admission of members, a fellowship in our College will become in time not only the sign of ability, but an introduction to business and reputation in physic."

In more modern days, aside from its monthly meetings for the presentation of papers and their discussion, certain Fellows have, with the consent of the College, established so-called "Sections," devoted to Ophthalmology, the History of Medicine, Sanitation, General Medicine, Laryngology and Otology, in which contributions are made to these special subjects.

From its earliest times this College has, when occasion demanded, acted as a guide or mentor in directing measures for the health and well-being of the city, state and nation. This is shown by the following quotation from "An Account of the College of Physicians of Philadelphia," written in 1912 by a former president, Dr. de Schweinitz. He wrote: "During the earlier years of its existence the activities of the College pertaining to the maintenance of public health and the improvement of public morals were noteworthy. For example, its appointment in 1787 of a committee to submit plans for establishing cold and hot baths and a botanical garden for the city; its consultation with the State Legislature over the spread of infectious diseases in 1789; its consideration in 1794 of "the regulation of the practice of physic within the State," and of the establishment of a quarantine and a hospital for contagious diseases at the request of the various authorities concerned; its work in conjunction with the Board of Health of the city in the management of the epidemic of cholera in 1788; its memorialization of the State Legislature in 1787 and of the Congress of the United States in 1790 on the deleterious effect of the excessive use of alcohol on the human system and the need of laws regulating its consumption; and its important actions in relation to the preparation of the Pharmacopœia of the United States begun in 1788. In each decennial revision of this work the College has taken an active part."

The library of the College is among the first in the world because of its collection if incunabula, and in this country is only second

to that of the Surgeon-General's Office in Washington so far as size and completeness are concerned. It contains 140,517 volumes and 139,394 unbound pamphlets, not including duplicates. Its incunabula is priceless and it has done much to make Philadelphia the source of the majority of medical books published in this country. It is open to everyone for study and research. So, too, is the excellent Mütter Museum, devoted to the illustration of medical and surgical subjects. Various lectureships have also been endowed by generous Fellows or friends of the College.

These statements are made, not in the spirit of boasting, but that young medical men may know how an institution founded by our forefathers has acted and does act as a center of learning, a source of knowledge and a standard of what may be designated as medical propriety. As a great cathedral stands as a symbol of religion, in the precincts of which everything is sacred, so the College of Physicians exists for no selfish purpose, and in its halls the intent is that its atmosphere shall carry a sense of the dignity of our calling, an honesty of purpose, a devotion to duty which must exist if the medical profession is to be in the future what it has been in the past.

I would ask you to remember that when one professes to be a physician he makes an avowal of a purpose not alone to earn a living but to serve mankind, that is, in the words placed by our Founders in the Constitution, "to lessen human misery." As the priest takes his vow to serve God, so should the physician take a vow to serve mankind. Here in a temple of medical learning may young men find the inspiration to love truth for its own sake and take away from its portals a sense of the dignity of their calling and a ceaseless desire to increase their knowledge, that they may render the true service that the traditions of the profession demand.

"*Tempora mutantur et nos mutamur in illis,*" the "times change and we change with them," in the sense that we adjust ourselves to the outside world; but in these halls this Latin motto fails, for the ideals that animated the Founders are ideals that are unchangeable because they are fundamental. If your visit to this College

now and in after years serves to impress you with a sense of obligation to advance medical science, to be worthy of the regard of your medical colleagues, to put aside all temptation to disregard the golden rule, the labors of our fathers in medicine will not have been in vain and the Fellowship as it exists today will receive encouragement to "carry on" the tasks which in future will fall into your hands here, or elsewhere if your fortunes take you far afield.

The Profit and Loss Account of Modern Medicine

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My subject is "The Profit and Loss Account of Modern Medicine." Wonderful progress has been made in medicine during recent years, but the profit has been attended by a loss which must be considered in balancing the account. In taking stock of the gain, we will find inspiration for the future; in counting the cost, we may guard against the undue sacrifices of the past.

The most distinct profit and loss are seen in medical education. More than a decade ago it was recognized that an increasing number of low-grade practitioners were being graduated each year by medical colleges and licensed by state governments. An investigation of the medical schools showed that many of them were poorly equipped, had scant clinical material and lacked sufficient funds to secure the necessary time of efficient teachers. A deliberate and systematic movement was inaugurated to remedy this evil. By moral suasion, by state legislation and by the combined efforts of the better schools the entrance requirements were advanced, the number and length of the teaching sessions were increased, the character and scope of the curricula were improved and the minimum number and approximate pay of the full-time teachers were specified.

The result of this propaganda has been that the total number of medical schools in the United States has been reduced from 160 to 80 and the total number of medical students from 28,142 to 17,432. In other words, 80 medical schools weak either educationally or financially have ceased to teach and over 10,000 medical students not properly qualified for the profession have ceased to study. And this despite the increase in the population of the country, which has occurred during the period covered.

The benefit of this movement is already markedly seen in the medical colleges, where the qualifications of the student are found improved and the character of the instruction more satisfactory. A medical student now begins his studies with a knowledge of the elementary sciences. He is taught the fundamental medical facts in the laboratory and dissecting hall by trained instructors who give their entire time to the work. When he comes to study disease it is in people, not in books. He is brought to the bedside of the patient and encouraged to observe, to weigh and to decide. The new idea in teaching tends to make of the student an active investigator instead of a passive hearer, and of the teacher a sympathetic guide rather than a verbose expositor.

There has not yet been time for the benefit of the change to be very apparent in medical practice, but the lessened number and improved quality of the graduates turned out each year will unquestionably in the end result in a great improvement in the ethics and efficiency of the profession.

The profit, however, has not been without its loss, and while we congratulate ourselves on what has been gained by this educational movement, it is only just to count what it has cost. Many worthy although struggling colleges have been put out of existence and their property practically confiscated, and many earnest and promising young men have been denied an opportunity to study medicine because of some defect in their preliminary high school or college education. Again, the modern medical school is not self-supporting and is a heavy financial tax on public funds or private philanthropy. Expensive laboratories, salaries of full-

time instructors and the necessary provisions for clinical teaching impose a cost that can never again be met by tuition fees. If each student were charged what it actually costs to teach him, none but the rich could afford to study medicine. The rich, as a rule, do not care to become doctors, and as doctors are not a luxury but a necessity, then the rich will have to be educated to contribute of their wealth to make doctors. Medical education has ceased to be a business and become a philanthropic work which must be supported by state appropriations and individual benefactions.

Finally, the cost of the modern method of teaching is seen in the graduate himself. If he has not paid in money he has been made to pay in time for his education. He has been kept in laboratory, lecture hall and hospital ward a non-producer, dependent on others for his support, until he reaches an age at which most of his contemporaries are married and settled in life. He is conscious of the sacrifice he has made and usually overappreciative of the attainments he has acquired. He desires to be a specialist and will only do general practice as a means to an end. He is determined to locate in a city and unwilling to settle in the country, preferring to starve himself in the one rather than to starve his ambitions in the other.

This results in an urban congestion and rural depletion of medical men which has reached a point to give serious concern and for which some remedy must be found. It has been proposed that special medical schools be operated to produce low-grade practitioners for country consumption, but this is impracticable and unthinkable.

The remedy for the evil is to make country practice less arduous and more profitable, and it is hoped that this will come about in time with the evolution of our social, economic and political life. When we compare the conditions that exist in the country today with those which existed even twenty years ago, and recall the changes that have resulted from the good-roads movement, the development of the automobile, the installation of telephones,

the introduction of parcel-post and rural-mail delivery, the improvement of the public-school system, the perfection of heating and lighting plants and the invention of labor-saving machinery, it is not difficult to believe that in the not far distant future the increased population of the country, due to emigration of health- and pleasure-seekers from the city, and the increased prosperity of the country, due to intensified and scientific farming, will make the life of the country doctor one that will attract and hold the best representatives of the profession.

Having considered the profit-and-loss account of modern medical education, we now come to what we have gained and lost in the doctor himself. The physician of the old school was usually a gentleman by birth and breeding. He was given a classical education, not because of his future profession, but because it was a privilege accorded his brothers as well as himself, without reference to their future vocations in life. His preparation for practice consisted in reading medicine for a few months in a preceptor's office and then attending lectures for one or two years at a medical college. His very lack of technical training gave him independence and resourcefulness, and with experience he gained an ability to make a diagnosis by intuition and to apply treatment which, while often empirical, was usually effective. He was no specialist, but attended every member of a household, because a family was a unit and his art was catholic. He knew the constitution of his patient, because neighbor married neighbor and lived where they were born. He was not only physician, but friend, confidant and counsellor as well. In his personal affairs he was unbusinesslike, rarely sending bills and accepting such honoraria as were tendered him in settlement of his accounts. In public affairs he was prominent and his views and opinions had weight in matters of church and state. He had his weaknesses and his faults. Measured by modern standards, he was ignorant and sometimes mischievous, but he served well his day and generation and was a most lovable old aristocrat.

The modern medical man begins to be trained for his profession

while yet a boy. His preliminary education in high school and college is scientific rather than classical and gives him knowledge rather than culture. When he completes his four years' course in a medical college and one year postgraduate work in a hospital he represents an investment of time and money covering a period of from fourteen to sixteen years. He is no longer a boy, but an eminently practical man, and he regards his calling as more a business than a profession. He recognizes the fact that he lives in an age of specialization, that no one man can now meet all the professional needs of a patient, and that the day of the domination of the family physician, on the one hand, and the dependence of the family, on the other, has passed. He understands that with the freedom now customary of choosing different attendants to treat separate ailments, the factors of social position, family connection and even personal friendship count for little, but that a doctor is employed because he is believed to be the most efficient man available to relieve the patient or cure the disease. He recognizes the necessity of sobriety, industry, honesty and clean living, but he also knows that the public no longer measures experience by age, virtue by matrimony or morality by affiliation with the church, and that the first and last prerequisite for success is professional ability.

Such being the situation with which he has to deal, the modern medical man early chooses a special line of work and devotes every effort and utilizes every opportunity to perfect himself and impress the community with his proficiency. His attitude to the public has changed and he no longer cloaks his reasons in secrecy or his actions in mystery, but deals frankly with his patients, explains cause and effect and secures their coöperation in carrying out treatment. The modern doctor has discarded the silk hat and frock coat of his predecessor and put on the sack suit of the business man. In a sense, he has become commercial. His offices are not only provided with instruments of diagnostic precision, but also with modern methods of keeping accounts and collecting fees.

From the foregoing crude pen picture must be inferred what has been the profit and what the loss to the public and to the profession from the modern doctor. We have lost a character dear to literature and gained a type perhaps less ethical and more mercenary, but which is certainly a scientific instrument of greater professional efficiency.

The development of the modern specialist is a source of both profit and loss to medicine. The profit is too apparent to need emphasis. The specialist, by concentration of study and limitation of practice to certain definite organs or diseases, is able in a few years to acquire greater diagnostic skill and more successful methods of treatment in his special line of work than another man of equal ability would obtain in a lifetime of general practice.

The presence of a specialist in a community not only gives to patients suffering with certain diseases opportunities for efficient treatment, but also offers to the surgeon and general practitioner a consultant whose opinion and advice are often invaluable. While the profit side of the specialist's account is large, still on the opposite page we find some items of loss. The high esteem in which the specialist is held and the pecuniary rewards which his services command have made him a victim of imitators and impostors, both inside and outside the pale of the profession. Modern medicine is not responsible for the quacks and charlatans, but it is responsible for the members of the regular profession found in every town and city who claim to be specialists, but who really do a general practice, and for others who, while they may limit their work to certain diseases, are not qualified as experts, and have no more knowledge or experience than the average general practitioner.

Again, the specialist, although an expert, is often narrow in his views and prejudiced in his opinions, so that he finds explanations for every symptom in the derangement of the organs he treats. His patients often suffer from special attention and general neglect. Motes are pulled out of the eyes and beams are left in the belly, or the abdomen is invaded for real or supposed appen-

dicitis and the lungs are left to fight their own battle with tuberculosis.

Finally, the specialist is an expensive friend of both the patient and the general profession. It is an everyday experience for the surgeon or general practitioner to send an obscure case, first to the pathologist for the examination of his blood, urine, sputum, feces or stomach contents, then perhaps in turn to the roentgenologist, the cystoscopist, the ophthalmologist and the dermatologist. The patient goes the rounds submitting his anatomy and functions to exhaustive examinations and reports and his pocketbook to depletion. The system is not essentially wrong. Unquestionably the patient is better cared for than formerly. To the well-to-do, while the cost is great, it is not prohibitory. To the poor the public and private charities are open where they can get the same services dispensed in less luxurious fashion but no less efficient. The real sufferer is the great middle class. Caught between penury and pride, without the price to pay but with the desire to conceal their poverty, they are often limited to an inferior grade of service.

Team work is essential to carry out the modern system of examinations. It is best seen in the staff of a modern dispensary or hospital where every patient has at his command the services of specialists in all diagnostic lines. The same principle must sooner or later come into vogue in private practice. Ultimately doctors will have their offices in large buildings instead of at their private residences. Here composite groups will unite in close business and professional association as a firm or corporation. Patients will be treated jointly, conveniently and expeditiously, and a combined bill will be presented through a central office.

One of the most wonderful gains made in modern medicine is in the exact diagnosis of disease by laboratory methods. For a time our knowledge of etiology and pathology was vague and indefinite, but one after another great discoverers have cleared the field and given us definite facts with which to work. Diatheses and dyscrasias, miasmatic and idiopathic diseases, are no longer

mentioned; the terms scrofula, blood poison and typhomalarial fever are no longer employed, and even the identity of neurasthenia and autointoxication are questioned.

We now diagnose the existence of tuberculosis, not by hectic fever, but by the demonstration of Koch's bacillus. We diagnose malaria, not by the therapeutic test of quinine, but by the presence of the plasmodium of Laveran. Widal has given us the agglutination test for typhoid and Wassermann the reaction which shows the presence or absence of syphilis. The white blood-count tells the degree of infection and resistance of the patient, and is not only a test of importance in making prognosis, but often indicates the proper time for intervention. The microscopic examination of tissue differentiates benign from malignant tumors, and in operation for cancer the frozen section will often tell the surgeon when he has reached the limit of the disease. The *x*-ray shows the existence of fractures and the position of fragments, locates the presence of stones in the kidney, ureter or bladder, and by perfection of technic makes pictures showing the passage of food from the stomach to the rectum.

The inspection of the modern laboratory is impressive to the visitor. The rows of reagents, retorts and test tubes; the microscopes, centrifuges and microtomes; the refrigerators, incubators and culture media; the polariscopes, hematocytometers, sphygmomanometers and other instruments of precision—make a layman and even some of the profession think that the work done and the final report made must settle all questions in a given case.

But the laboratory method of diagnosis entails a loss as well as a gain, and has its dangers and disadvantages. While it is true that chemical reactions are always constant, that the microscopic field shows the cellular structure of tissue and the physical form of bacteria, and that the *x*-ray picture truly depicts the shadow of the object between the Crooke's tube and the photographic plate, it must always be remembered that there is a personal and uncertain factor in the result, namely, the laboratory man who

construes what he sees. A poor pathologist or roentgenologist is worse than none at all, and even the opinion of the most experienced and proficient is occasionally wrong.

As valuable as are his services, the laboratory man is sometimes too highly regarded. Seated upon his kingly stool and surrounded by a rarefied scientific atmosphere, he tends to tyrannize the clinician. His reports are too often accepted as final in their decrees and become enervating in their influence. Owing to a tendency to lean too much on laboratory reports, case histories and bedside records the profession is in danger of neglecting the examination of the patient. Sick people are just as instructive today as in the time of Sydenham, Addison and Bright. Laboratory data and clinical findings must be studied together. They must be compared, and one used to check a possible error of the other.

The hospital, while an old institution, is modern in its distribution and function. It has lived down its stigma of a death house. It has overcome the prejudices of the masses and appealed to the pride of the classes. Practically every town of five thousand inhabitants has a hospital, and every well-regulated hospital is an asset to its community. A hospital is now accepted as the safest, most comfortable and most economical place for the seriously sick, and it is also recognized as a local center for the dissemination of knowledge among the public, the training and education of nurses and the uplift of the profession by the demands made for good records, thorough examinations, accurate diagnoses and rational treatment.

The modern hospital, however, is not without its dangers and disadvantages, as it offers opportunity and hence temptation to members of its staff, especially those with surgical ambition, to undertake work for which they are not qualified. The following is a familiar illustration: A small town feels the need of a hospital. The women organize, raise the money and build one. The people of this community had formerly made it a practice to go to some neighboring city when in need of special medical

treatment or a serious surgical operation. They are now urged to patronize the home hospital, and as that course appeals to their desire to help a local institution, and also avoids separation from family and friends, the advice is often followed. For a time an experienced surgeon is sent for to operate on difficult cases and one of the local practitioners acts as his assistant. The successful result which usually follows in these early cases inspires the community with confidence in the hospital and in time creates a desire in the mind of the local man to do the work himself. He spends six weeks or three months at a postgraduate school and returns with a highly embellished certificate. He performs a herniotomy or removes an appendix and the patient does not die. He comes to be known in the community as a man of wonderful nerve. He hopes later to drop his other work and do nothing but surgery.

The evil goes further. This newly developed surgeon has no regular assistant and makes it a rule to get the family doctor of the patient to help him with the operation. As the physician does part of the work it seems only proper that he should get part of the fee. When this practitioner has a patient who desires to go to a specialist in a large city, what is more natural than that he should go with him and explain that he was reluctant to come because of the loss of the financial benefit he was accustomed to receive from such cases at home; or what more human than for the city surgeon to endeavor to meet this competition by offering to split the fee in this and future cases, provided it was made sufficiently large; and what more necessary than that this secret understanding between the two be kept from the knowledge of the patient. And so has come about the great modern evil of the secret division of the fee, a practice by which the doctor sells the patient to the highest bidder, and by which the surgeon robs the patient to pay the doctor.

The evil of incompetency in the shape of the unqualified surgeon and the vice of dishonesty in the form of the secret division of the fee are being fought in the profession by the establishment of a society known as "The American College of Surgeons." If the

remedy is not found, action will be taken sooner or later by an indignant public through state legislation.

The advent of the trained nurse marked an epoch in medicine almost equal to the introduction of anesthesia and antiseptics, and the name of the founder of the order, Florence Nightingale, deserves rank with those of Long, Pasteur and Lister. The rapid and general adoption of the trained nurse was due not only to the professional needs of the doctor but also to the domestic necessities of the public. In times past a sick person was nursed by servants and relatives. In every family there were old mammies and old maids who had considerable practical experience in nursing and who derived a certain morbid pleasure from the temporary authority of the sick room. The modern servant problem and the recent migration of the unmarried female members of the family from the home to the office did away with the supply of amateur nurses and created a demand for the professional nurse.

At one time there were but two respectable things for a young woman to do—get married or teach school. Now, many avenues are open to them, and of these none is more attractive or offers greater opportunities for service than the field of nursing.

Time will not permit an attempt to show the contributions of the trained nurse to the progress of medicine. In every department she has proved a faithful, efficient and trusted worker, without whose aid the end attained could not have been accomplished. At the bedside of the patient in the silent vigils of the night, in the operating room during the stress and strain of nerve-racking ordeals, and recently in Europe, on battlefields torn with shell and red with carnage, she has shown a courage, a fixity of purpose and a devotion to duty rarely equalled in either sex or in any profession.

The nurse is a woman and therefore has her faults, but the faults are those of the woman, not of the nurse. If she is sometimes spoiled, occasionally a trifle tyrannical, and more rarely a bit supercilious from real or supposed superiority of knowledge, it shows the weakness of her sex and not of her profession. If her

services are sometimes prostituted to pamper the whims of the neurasthenic invalid or to indulge the selfish indolence of the idle rich, it is not her fault but the fault of our present system of living.

The medical society is an important factor in the progress and development of medicine. While some members of the profession do not appreciate the advantages to be derived from regular attendance and active coöperation in medical organizations, it is a fact that the busy and successful practitioners are usually present at all the meetings of their county, state and national associations. This can only be explained by the fact that those of the profession whose experience and judgment have proved to be the soundest believe that medical meetings are profitable.

Medical societies usually hold their regular sessions in different cities, and their meetings educate and stimulate the local profession and advertise to the laity the fact that medicine is not bound by dicta and dogmas, but is a progressive science ready to discard the old if it is proved to be fallacious, and to adopt the new if it is found to be of value. The meeting of a medical society enables its members to read papers, thus giving them a legitimate opportunity to show their capacity and to present new and original views as to the treatment of disease, thus adding to the knowledge of the profession. It enables its members to hear papers read by others, thus giving them an opportunity to gain an amount of information they could get in no other way with so little labor and in so short a length of time. The discussions that follow these papers are especially profitable. In them is an impressive personal element that is totally lost in the stenographic report published in the *Transactions*.

In addition to the educational and professional advantages derived from these meetings there are equally important social and personal benefits. The occasion is a holiday, a recreation, a vacation. It breaks the monotony of life and enables a man to do better work when he returns home. It offers the opportunity to meet men who are doing the same kind of work in different sections

of the country, and results in pleasing and profitable acquaintanceships which often lead to permanent friendships. And last but not least, it brings together men who live in the same community, but who, owing to petty jealousies or lack of time for social intercourse, see little of each other. Either in the session of the society or in the committee room or on the journey to and from the place of meeting, they are thrown into an intimate contact which frequently leads to explanation of misunderstandings, adjustment of differences, appreciation of good qualities and to the establishment of the most friendly and cordial relations.

Despite the manifest and manifold benefits of the medical society it is necessary from the profit to deduct a loss. There are many medical societies which have no right of being and have been organized simply for political or personal motives. Originally established to give office or secure patronage for a certain group of men, they are often supported for years through a mistaken pride or patriotism on the part of their members, to the great injury of the legitimate societies whose territory they cover. There is need for a movement to standardize medical associations, and until this is done through the proper channel the profession should try to minimize the evil by withdrawing its membership from superfluous organizations.

Twenty years ago it was considered derogatory to the dignity of one clinician to visit the workshop of another. To do so would be to invite the criticism of a confessed inferiority or of a desire to spy on the work of a competitor. Then all one practitioner knew of the work of the other was through printed matter, and some things that were true were not believed and some things that were not true were accepted. It has now become almost a custom for the busy surgeon and physician to devote two or three weeks of the period previously assigned for a vacation to the duty of seeing, at first hand, what his fellows are doing. At the various recognized medical centers every provision is made for the convenience and instruction of visiting doctors. No fees are charged and the veriest stranger is made to feel welcome. By actually observing the

methods practised in these various clinics the visitor is able to decide whether or not they are preferable to the technic he had hitherto employed.

Surgeons, as a rule, attend clinics more frequently than do physicians. This is a pity, for even in a strictly surgical clinic the points of greatest interest are not the methods of operating, but the explanation of symptoms by the pathological conditions found. It is a curious fact that many physicians who would travel miles to see a postmortem examination will not go around the corner to witness an operation which demonstrates the same changes produced by disease in living tissues before they are obscured by terminal results. To correct the loss entailed by the failure of the average physician to avail himself fully of the advantages offered by the modern clinic, the internist must learn that to keep abreast of the times it is necessary not only to study but to travel as well.

For centuries the world suffered from pestilence and scourge. People in desperation abandoned themselves to their fate, discarded even the crude laws of the Mosaic Code and attributed their condition to the visitation of the devil or the wrath of an offended deity. But medical men have ever labored to discover the cause of disease and the means to prevent its occurrence. With an altruism rarely approached in other professions the physicians of the past and the present have given unceasingly of their time and labor to destroy the very source of their living.

Time will not permit a record here of the victories that have been achieved in preventive medicines, beginning with smallpox and now approaching a successful issue in the case of yellow fever, malaria, typhoid and other diseases. The completion of the Panama Canal, a task rendered possible only by the sanitary regulation promulgated and enforced by Surgeon-General Gorgas, stands as an unquestioned tribute and enduring monument to the perfection and efficiency of the measures which modern science has developed for the maintenance of health and the prevention of disease.

The Public Health Service is now recognized as one of the most

important departments of our general and local government. The work in this service offers the present-day graduate one of the most attractive fields open to him. It does not hold out the promise of fortune that goes with rare eminence in private practice, but it guarantees to every worker a reasonable income, the opportunity for scientific study and research, the certainty of performing a useful service for his community and the possibility of becoming a great benefactor to the human race.

Public health work marked the beginning of a new era in the relations between the profession and the public. It was characterized by an effort on the part of the profession to take the public into its confidence. Its purpose was to make the people a partner in the conservation of health. A short time ago, if a doctor addressed a lay audience on a medical subject his motives were questioned. Now the profession employs every agency of publicity to spread the propaganda against disease. The columns of the newspapers and magazines, the walls of public conveyances, the lecture platform, the pulpit, the school and the drama warn and plead against the danger of the mosquito and house-fly, the communicability of tuberculosis, the insidiousness of cancer and the pathos of "damaged goods."

Publicity in medical matters has undoubtedly done good, but it has also done harm, and here as elsewhere we must record not only the profit but the loss.

The first loss is seen in the schools, for an examination of the text-books employed in physiology and hygiene will show that just as at one time children were taught false history, so now they are often taught false science. Another evil is the attempt to teach sex hygiene. It is a difficult question to decide in an individual case when and how to impart this delicate information. If parents hesitate to discuss the matter with their child at home, it is certainly an evasion of responsibility attended by great danger to turn the subject over to an old maid teacher to deal with in a mixed school.

Finally, the general information given the laity on medical sub-

jects has caused a certain indefinable loss to women by an impairment of the charm of innocence. Matters are now discussed in a mixed audience with a freedom and frankness that would have been thought unbelievable a generation ago. Beginning with coeducation and equal suffrage, subjects suggestive of sex differences, the field of activity of the female mind has broadened until now the average high-school girl is more or less familiar with the problems embraced under the terms eugenics, race suicide, the social evil, the age of consent, the white-slave traffic and the regulation of the red-light district.

The woman of today has lost her prudery. Let her beware lest she lose her modesty as well! If such should prove the case, it would be necessary to change from the credit to the debit side the balance now found in "The Profit and Loss Account of Modern Medicine."

PASTEUR

Born December 27, 1822

ADDRESS

By ETIÈNE BURNET, M.D.
SOUS DIRECTEUR DE L'INSTITUT PASTEUR DE TUNIS

[On the evening of January 2, 1923, the College of Physicians tendered a reception to Dr. Etiène Burnet, of the Pasteur Institute at Tunis, the official delegate of the French Government to a celebration which was held in Philadelphia on December 27, 1922, in honor of the centenary of Louis Pasteur.

Preceding the reception the invited guests and Fellows of the College assembled in Mitchell Hall, where, after being introduced by the President, Dr. Thomas R. Neilson, Dr. Burnet delivered the address which follows.]

It was in the order of things that the celebrated College of Physicians of Philadelphia savants should celebrate Pasteur's hundredth birthday with enthusiasm. Not only have you stood among his earliest disciples, but in no country has his teaching been more fruitful than in this one; nowhere have the Pasteurian spirit and technic more efficiently influenced the features and development of civilization. Supposing he happened, through some miracle, to be present among you, he would receive your acclamations, to return them to science, but he would, moreover, convey to you his own gratitude, because the improvement of mankind through the means of scientific hygiene has become one of the fundamental tenets of what we call Americanism.

I believe that in no other country in the world would not only the élite but the bulk of the nation be more apt to understand the great personality of Pasteur: The will that knows of no

obstacles, unwearied industry and self-modesty in the midst of the most signal triumphs, the sentiment that duty lies first of all in altruism and devotion to mankind.

Nowhere would he meet with the very idea of humanity more alive, of humanity, that is to say, of the organized collaboration of all peoples, with a nobler and happier life for their end and science and peace for their means.

Pasteur belongs to all mankind. Therefore, the celebration of his birthday is in every country in the world, today, an international festival of science and peace.

How shall one speak of Pasteur on such an occasion? All has been said about him. Men who lived for years with him and had a share in his work, have written remarkable books on him, which are in everybody's hands. His discoveries and experiments belong to the every-day routine of every laboratory. For the chemists, physiologists, biologists, agronomists, hygienists, surgeons and physicians here assembled, nothing can be said that would be fresh matter.

Still, we feel there is something to be said.

Since, in the course of our research work, we constantly get opportunities of admiring him in detail, with the short-sighted eye of the scientist, to scrutinize his work, so to say, through the microscope, our emotion, today, will rather take a plain and full view of him. Some twenty years ago, every opportunity was rightly seized upon to expound his work afresh. Now that every scientist has assimilated it for himself, let us rather view it from farther back. Let us look at him as he will be looked at in a couple of centuries, as we ourselves look at Lavoisier, Galileo, Kepler. We shall see his work rise still higher, in the same way we see a mountain rise as we step farther back, and tower over all his age, such as we see Mount Blanc overtowering the Alpine range when we look at it from a distance.

On the other hand, since we wish to evoke today not only his work, taken apart from himself, but his personality, we should

like to show him full life, mighty and good, such as he was. Just as most excellent Plutarch, after a lapse of several centuries, would bring again to life the illustrious men of Greece and Rome in those simple biographies which have helped to the making of so many great spirits, so one ought to show Pasteur, simplified and legendary, such as he is shown this very week to the children in all the schools of his own country that his example may rouse vocations and devotions, and such as he appears to ourselves when we fear to break down before difficulties, and he of all others becomes for us the prompting spirit.

Posterity says of him, first of all, that he made medicine into a positive science.

He was not alone, indeed, to work at the bringing about of this transformation. To mention only the greatest among his contemporaries, the name of Claude Bernard is likewise inseparable from experimental medicine. But, as Pasteur made out the cause of the great diseases of man and domestic animals; as he created remedies through his discoveries on vaccinations; as, by the treatment of rabies, he triumphed over a disease which, notwithstanding the unimportant space it held in statistics, had a dreadful influence over the mind—Pasteur's demonstrations gave the most rapid and decisive impetus to medicine.

This historical fact is the more astonishing as, while Claude Bernard had from the outset begun with the study of living organisms, Pasteur had begun with the study of inert matter. Pasteur was a chemist. Nay, when yet a very young scientist, he had come to the fore through a signal discovery in the field of crystallography: He was so fond of crystallography that to the end of his career, he regretted to have been under the necessity of relinquishing that study, and after the discovery of vaccine, he would still move the hearts of his disciples when talking of crystals and of the dissymmetrical forces in Nature. Pasteur was a chemist in a time that was in our country the golden age of chemistry. He was a precise manipulator who could work skil-

fully on traces of matter, and he applied the chemist's technic to the study of ferments and diseases.

The renovation of medicine by a laboratory scientist, by a non-physician, however paradoxical though utterly logical it appeared, was remarkably instrumental in the converting of general opinion to scientific methods in connection with the study of life.

Pasteur's struggles against the physicians, or rather against the medical spirit of his time, are well known. No word can better sum them up than those Pasteur addressed to a physician who was repeating, in 1885 (!), objections that had been hundreds of times refuted for the last thirty years:

"Sir, I don't understand much of your language. I am no physician, and at times I don't wish to be one. I am a chemist. I make experiments and endeavor to make out what they purport."

Once we have acknowledged that Pasteur also met with enthusiastic supporters among the physicians of his time, and that today's medical profession is, as I imagine, wholly converted, we may well be permitted to have a laugh *en passant* at the physicians of yore. May be such a man as Pidoux owes what little celebrity he still enjoys to such dicta as the following:

"It is not within the power of physiology to account for the simplest morbid affection."

"Tuberculosis is the general outcome of a multitude of sundry causes, both internal and external, and not the achievement of some specific agent always the same. Specificity immobilizes medicine."

"Disease is within us, from us and through us."

"The new doctrines, when applied to chronic diseases, bind us to search for specific remedies, or vaccines, in consequence of which all progress is checked."

In that time a surgeon, who was a man of some skill, would speak before the Academy of Medicine of Paris of "that laboratory surgery which kills many animals and saves very few men."

Piorry, in 1877, would exclaim, "It is not the disease, an abstract thing, we have to treat; it is the patient."

"One aims at the microbe and one brings down the patient," another could say.

Toward 1883, Peter spoke of the new conquest as follows:

"Those are curiosities of natural history which, however interesting they may be, are of little or no avail to medicine proper, and are worth neither the time spent on them nor the noise made about them. After so many laborious researches, nothing will be changed in medicine—there will only be a few more microbes."

The same Peter uttered this sentence, "Microbicidal medicines—homicidal medicines."

Léon le Fort, a surgeon, said at the Academy of Medicine:

"I believe in the interiority of the principle of purulent infection with certain patients; that is why I am against extending the theory of germs to surgery, which proclaims the constant externality of the said principle."

And to wind up with chemistry, from which we started, let us quote this decisive dictum from Prof. Peter:

"M. Pasteur's excuse is that of being a chemist who wanted to reform medicine, to which he was an absolute stranger."

And what was the state of things in surgery before Pasteur's era? My great master, Elie Metchnikoff, had gathered from statistics of the war in the Crimea (1854-56) the following figures: Among the men amputated of a leg, 92 per cent died. The amputation of a thigh caused 71 per cent casualties.

As to femoral fractures, it was a better lot not to be operated upon. Among the non-operated men there were 68 per cent deaths; among the operated ones there were 92 per cent deaths.

Then the Russian surgeon Pirogoff believed in some mechanical alterations that easily turned into chemical alterations, and Lister at that time believed that oxygen was causing the scourge.

While he was turning medicine into a positive science, Pasteur was founding scientific hygiene. Preventive hygiene with regard to infectious diseases is wholly contained in the theory of germs and the invention of vaccines. This hygiene does not appear

upon the world's stage as a humble complement of medicine, but as part and parcel of it. Nay, hygiene *is* medicine, the new medicine, which is to supersede the old one.

The field of hygiene has been infinitely extended until it embraces individual and social life. Pasteur it was who created and, so to say, launched it.

Man readily welcomes only such ideas as foster his interests, and he is first of all interested in his own health and life. When Pasteur brought under the control of science those goods to which man was by instinct most attached, he led him into the habit of allowing his whole life to come under that control.

Pasteurian medicine and hygiene have more than any other influence helped to the scientific education of the masses.

One has not been without saying that without Pasteur this evolution would have been achieved all the same, and medicine would have found its way. That may be. But with Pasteur the evolution was more rapid, and it was more beautiful, which also is not without importance. Thanks to Pasteur, mankind has gained God knows how many scores of years, and ever so many millions of human lives have been spared for progress. This is the very definition of the man of genius. Besides, we owe it to Pasteur to be granted the intellectual joy of tracing the course of that advance in the progress of one and the same mind, whose logic and harmony fill us with confidence in the power of the human mind and the future of mankind; for it is beyond any doubt that Pasteur did not advance at random, nor even desultorily, and that he was at every step utterly conscious of his gains as well as of his aim. From the moment when he was poring over his crystals, he saw a new opening in the study of fermentations. As soon as he approached fermentations, he saw the infectious diseases: When he presented the Emperor Napoleon III with the book that contained his researches on wines, he told him that his experiments could reveal to him the causes of diseases. As soon as he has approached the germs, he saw the vaccinations.

No one has better helped to popularize that noble idea of the modern world that scientists are its prophets.

The next thing to be said of Pasteur is this: That he has, more than anyone else, confirmed the sovereign power of experiment, has set experiment above everything and has been the greatest master of experimentation. This can be truly said without abating anything of the admiration due to Claude Bernard and others. In the sciences of life, Pasteur's experiments are grand among the grandest because of their uniting to the utmost degree simplicity and fruitfulness.

Pasteur will concentrate a question, however extensive, into, as far as possible, one experiment, which he frees of all its unessentials, which he improves with a view to simplicity until it can be understood by the very child, and is proof against critics because it does not allow of any point on which criticism should be able to apply itself. He focusses all his hypotheses upon one chosen experiment, in the same way as a playwright will gather up the threads of his plots into one decisive situation, which is the crisis.

Dr. Roux, his collaborator in the researches on anthrax and rabies, writes as follows: "Pasteur would expound the most profound and unexpected ideas and propose the boldest experiments. That precise experimentator had a powerful imagination: Nothing was *a priori* absurd to him. But the most fiery impulses would always lead him to some experiments to be tried, and he would finally retain nothing but what stood proven."

The typical Pasteurian experiment, that which will ever be quoted, is that in which the whole question of spontaneous generation is concentrated: The experiment of the swan-neck balloon; or, again, the one that solved the question of the etiology of anthrax when the soluble virus was opposed to the bacillus, namely, the dilution of one drop of culture from one balloon to another, until one realizes a dilution in a volume superior to that of the earth, a dilution larger than any given quantity and still virulent. It is impossible to go further in the way of proof and simplicity.

As instances of remarkably fruitful experiments, let us mention the wrapper he applies on the grapes of a vine when seeking the origin of yeasts. This is the sanitary barrier of international hygiene; the sorting of the eggs or "seed" in the disease of silk-worms; this is the child that is isolated from the tuberculous family in order to save a generation.

One day Pasteur filters a culture of the microbes of fowl-cholera and finds that the inoculation of the filtrate, free from microbes, gives rise to all the symptoms of fowl-cholera. This was the origin of the researches on microbian toxins. One day he finds that the condition of an anthracic sheep improves after injections of killed Bacillus anthracis: This was the germ of bacteriotherapy.

Each of these experiments was like the creation of a world.

No one ever better showed how profound was this saying of Claude Bernard's, true in every science, but above all in biology and medicine, "The experimentator's power reaches beyond what he knows." Pasteur devises the prophylaxis of "pébrine" before he knows the nature of the parasites. He does not want to know the microbe of rabies—which is still unknown to this day—but to cultivate it in the nervous system of rabbits, attenuate or rarefy it in the spinal cord and turn it into vaccines. The Pasteurian vaccination against hydrophobia stands out as the masterpiece of experimental medicine.

If no scientist ever made more beautiful experiments, none either ever made a greater number of experiments of all kinds. We know that Pasteur would conceive and achieve such by the hundred, without ever heeding the unlikely or the impossible. No experiment, in his opinion, was impossible. There must always be found some means of realizing it. As Hunter said to Jenner, "Don't think, but try," Pasteur would say, "Well, try the experiment at once!" One stands wonderstruck at the perusal of his researches on wines, beer or silk-worms, and when one sees, by the records of his experiments, which he let nobody keep for him, how many trials it took him to come to results seemingly so simple as anthracic vaccines or antirabic treatment.

Imagination, divination of what must take place in Nature, Pasteur, we know, was possessed of that paramount gift. But plans, directions, motive power do not suffice for the piercing of a mountain; the boring machine wants a hard point to cut through the rock. Experiment is the boring point.

I believe that were some Emerson to add a savant to the series of his representative men, he would chose Pasteur, not only as a man of genius, but as the type of what may be called *scientific idealism*.

I am not only thinking of the private virtues, both domestic and social, which it is desirable to find in every man, even in the savant—above all in the savant, Pasteur would say—and which are not sufficient by themselves to make a great man.

What I mean is the attitude of the savant, as a man, before the great problems of human life, that particular wisdom that enables him to be rigorously a scientist, and, at the same time, not to let his soul wither and open it to generous feelings for lack of which human life would shrivel.

As a scientist, Pasteur admitted of nothing but facts and experience:

"The experimental method ought to be free from any metaphysical speculation."

Listen to what he said on the occasion of the polemic of so-called spontaneous generations:

"Religion, philosophy, atheism, materialism and spiritualism are quite out of the question here. I shall even go so far as saying, As a scientist, I don't care about these. Inquiries about the primary cause are beyond the scope of science. The latter acknowledges but what it can demonstrate, namely, facts, secondary causes and phenomena."

And again:

"Science ought not to pay any heed whatever to the philosophical consequences of its own labor."

"In each of us are two men: One, the man of science, and on the other hand the sensitive man, the man of tradition, of

faith, or doubt—the man of feeling. The two stand apart from each other, and woe to him who wants to make them encroach upon each other in the so imperfect state of human knowledge."

This is the attitude of every positive mind. It was Claude Bernard's attitude. Outside and above science, he and Pasteur professed the belief that pleased them: Pasteur, a more religious soul; Claude Bernard most probably a materialistic one. Never did their creeds interfere with their experiments.

But if, to take up again Pasteur's antithesis, the man of feeling ought not to influence the laboratory man, he, in fact, in any high-minded scientist, cannot help doing so, and exerting an influence of another kind, as a stimulant, as a source of energy.

The savant must have an ideal.

He cannot possibly minister to the evil powers. He cannot possibly pay any worship but to self-devotion, and peace among men and freedom. Bearing this in mind, we shall piously receive the lesson of Pasteur's life. He once said, at the unveiling of Thénard's, the chemist, statue:

"That share of the heart in the progress of science would be a fine one—one it would be useful to delineate."

One could make up a whole booklet, a real golden book, with the words by which Pasteur expressed his scientific idealism. Here are some of them:

"The cultivation of sciences in their highest expression is perhaps even more necessary to the moral condition of a nation than to its material prosperity."

"The labors of those addicted to things of the mind, in every department, raise the moral sense. Thanks to them, God's notion itself is spread abroad and exalted."

"The mind is leader in the world. Still, let us not forget that the way of great efforts is more often than not that of great sacrifices."

"As regards the good to be dealt out, duty stops short only where power fails."

Rabelais, the greatest writer of the French Renaissance, that

age when man was yearning for universal knowledge, once said: "Science without conscience is but the ruin of the soul."

For Pasteur, science must not bear obedience to any master, not even to conscience. But it is inspired by conscience.

We in Europe often speak, referring to that bounteousness with which, in America, acquired riches are made to serve great concerns of material and moral utility of business idealism. Pasteur would have also met with scientific idealism among you. Never has the world, as we all know, had such need of it. In an age when the technical toil of production is forced upon every man, life would get dull and barbarous did not man at the same time silently cultivate within himself moral inclinations and generous feelings. Society will not be habitable unless we are idealistic technicists.

Great men worship was one of the fundamental tenets of Pasteur's idealism. "Let us piously treasure up, he said, for the teaching of posterity, every simple word, every single act of theirs that can help to reveal the goads that urged their great souls."

I don't know whether Pasteur's letters will ever be published. They, more than anything else, would disclose "the goads that urged his great soul." But we are close enough to him and have enough testimony to hand to enable us to perceive the outstanding features of his personality.

He came of a peasant family. His mother is known to have been of a bright, imaginative nature. His father, a humble country tanner, was a hard worker. No wonder Pasteur was a modest, painstaking man: These are the staple virtues of common people who live close to the glebe.

Pasteur's father, moreover, had served in the years 1811-1815 in Napoleon's armies; whosoever knows, as the historian does, the mentality of those men will readily infer he had the faith and enthusiasm that animated the Revolution volunteers. Pasteur was born at a time when the bulk of the nation, freed by the Revolution, would enjoy their fresh liberty. Liberty, nation,

patrie—those words meant the same reality for them. This was still the time when *patriot* was still synonymous with Revolutionist. The idea that, henceforth, the nation is nothing but themselves, their lives, their possessions, their children, and then she must be maintained by their own efforts and devotion. Such is the idea that Pasteur, when a child, would daily hear from his father, and with which he remained imbued. I am sure that in America, likewise, such a time brought up such men.

So Pasteur was a hardworking man, as everybody was about him. He went on repeating: "Let us work! That is the only thing to amuse one."

But he did not only want to work, he also wanted to exalt himself through working, and a man of his condition, as he, along with his father, realized, exalts himself but through learning. When still a boy he wrote to his sisters, "With learning one rises above all others." While pretty far advanced in his studies already, he set to coaching his father, that the latter might, in his turn, teach his sisters; and at fifty-three the father is seen poring for two days over a problem of arithmetic, and he writes to the son, "When one has to learn with a view to teaching others, it is not trifle."

Not only was he a hardworking man, he was also a conscientious one. At all the periods of his life, at the time of his most engrossing researches, he would scrupulously acquit himself of the duties of his profession. While a young lecturer at the University of Strasbourg, and later at Lille University, he would give much time and many pains to the preparation of his lessons, and leave his laboratory for days in order to take his students to the factories of Flanders and Belgium.

He was by no means a precocious genius. Up to fifteen, he did not show any vocation save a taste for drawing. His ambition—his father's ambition for him—was a modest one; if he can only become a teacher at the small college of Arbois, the town where he has been brought up, he will have already marked the superiority gained through learning. Not before he is in Paris,

when he has come into touch with masters, does he get roused up: Then he will be a chemist; he is aware of his strength. The first discovery gives him the impulse. From that moment, he appears marvellously self-confident and audacious.

Purely intellectual gifts, such as versatility and quickness of the mind, the aptness to understand everything and dally with all ideas, these hardly belong to Pasteur. His are earnestness, insight, tenacity of purpose and incredible focussing of all his powers upon the subject on hand. He follows out his idea unswervingly and refers everything to it.

"His intent thought," says Dr. Roux, "would grasp difficulties and finally solve them, just as the blaze of the blowpipe, steadily blown on to a refractory body, melts it at length."

Hence those words, again and again repeated, the profound meaning of which is obvious to any laboratory man. Whenever he fails in a research, "Let us try again," he says. "Let us make the same experiment over again—the main thing is not to leave off the subject." When he has succeeded, he wants to proceed further: "You must not stop at the results you have attained."

Does anyone propose to associate to the research in progress a man who is working, for the present, at another question: "No, he says, let him alone, he is not in the subject."

Every question must be searched to the bottom:

"One should exhaust all combinations until the mind becomes unable of conceiving more."

"One should have seen Pasteur at his microscope," says Dr. Roux, "to get an idea of the patience with which he would examine a preparation. We often met him standing by a cage, motionless, indefatigably observing some particularly interesting guinea-pig or rabbit. He would survey the lots of animals that were being experimented upon with that sustained attention which nothing could escape; for whole hours he would watch a sheep which he suspected of being diseased."

To everything he would reply, as a sort of leit-motiv, "Let us work."

Does not this remind you of Lincoln's words, "I am always for the man who wishes to work."

When Pasteur told the students of Arbois College that he had come by his discoveries, to quote his own words, "through assiduous labor, without any special gift but steadiness of purpose, to which may be added, perhaps, the allurement of the lofty and the beautiful," he left aside, indeed, that unaccountable element, inventive genius; but he uttered that paramount truth, that the first and foremost qualification for experimental research is *character*, what Emerson described as "the power and courage to make a new road to new and better goals."

W. Ostwald, in his book on the great men of science, distinguishes them into two classes of temperaments, the classics and the romantics—the classics being those who go along a road drawn by them, in most cases, to develop an inspiration of their youth and follow it logically, deliberately, with every step made secure, with nothing left unfinished, the romantics being the men with varied flashes of inspiration, with a fiery imagination, who astonish their contemporaries to a higher degree, but are also in danger of petering out more rapidly. In which of these two classes shall Pasteur rank?

His inspirations are extraordinary, he renews himself, up to the end of his long career, without getting exhausted; and, on the other hand, he evolves his ideas to their furthermost consequences, makes his gains secure and leaves nothing imperfect or unfinished behind.

Pasteur is both a classic and a romantic. He has all gifts in one. He is the great thorough man of science.

Lastly, the commemoration of Pasteur's birth must be an international celebration of peace.

Pasteur, as we know, loved his country passionately. But who would dare suggest his patriotism was not a pacific and humanitarian one? We do not know of any utterance of his in which he separated his country's cause from that of mankind. Let us recall his speech at the Congress of Copenhagen (1883):

"By our presence at this Congress we affirm the neutrality of science.

"You will always find a great patriot in every scientist: The ambition of seeing the nation to which he belongs take or keep the first place urges him into the toilsome but glorious undertakings of learning which lead to the true and lasting conquests. Mankind then avails itself of those labors that come to it from every part. It compares, chooses proudly and takes possession of all the national glories."

Or, again, his speech at the Congress of Sericiculturists in Milan (1876):

"Science has no homeland, because learning is mankind's patrimony, the torch that enlightens the world. Science ought to be the highest embodiment of one's country, as, of all nations, that one will ever be the foremost that stands foremost by the works of the thought and the intelligence. I drink to the pacific contest of science."

In 1871, he sent back the doctor's diploma that had been granted him by the German University of Bonn. He sent it back, "not without protesting (such are the very words of his letter) his profound respect toward the Dean and all the illustrious professors who signed it." But why should one not quote, as well as this fact, the post scriptum of his letter to the Dean of Bonn, Dr. Naumann:

"And now, sir, when I read over again your letter and mine, I feel deeply grieved that men who, like you and me, have devoted their lives to the search for truth and to the progress of the human mind, should talk to each other in that way."

Those lines betray such sadness that they, more surely than anything else, convey, under those circumstances, the inmost thought of the man.

On the day of the inauguration of the Pasteur Institute, his whole thought is for humanity and peace:

"Two antagonistic laws, he says, seem to oppose each other nowadays: One, a law of blood and death which, by daily devis-

ing some new war implements, compels the nations to be ever ready for the battlefield; and the other, a law of peace, of work, of salvation, which thinks of nothing but delivering man from the scourges that besiege him. The one aims at nothing but brutal conquests; the other at nothing but the relief of mankind. Which of the two will gain the upper hand? Only God knows. But we can affirm this: That French science will have endeavored by obeying that law of humanity, to extend the bourne of life."

And, to wind up, on the day of his jubilee, in 1892, on the day when, glorified by the whole world, he utters his supreme thought, these are his words:

"You, delegates from foreign nations, who have come such a long way to give France a token of sympathy, you bring me the most profound joy that can be felt by a man who firmly believes that science and peace will triumph over ignorance and war, that the nations will come to an understanding with a view not to destroying but to edifying, and that the future will belong to those who have done most in behalf of suffering humanity."

One hundred years ago Europe was, as it is today, on the morrow of great wars. Pasteur's father had been campaigning both in Spain and in France. We cannot help thinking that some chance bullet would have been enough to prevent Pasteur from being born.

Today, when we think of the ten millions of men who have been killed in this World War, we are entitled to say: How many, among those who died, would have done something useful for mankind? How many bright and generous geniuses the war has destroyed or prevented from being born?

Therefore, Pasteur verily teaches us to believe and wish to our last that ignorance and war be put down by science and peace.

APPENDIX

PROCEEDINGS

OF THE

SECTION ON OPHTHALMOLOGY

JANUARY 18

Bilateral Optic Nerve Disease Following Sinus Infection

Dr. Luther C. Peter presented a man, aged thirty-five years, who suffered from bilateral uveitis in 1915, with a second attack in 1918. Laboratory studies failed to reveal anything but a positive von Pirquet, although the clinical examination seemed negative. An *x*-ray study showed old foci of disease in the apices of the lungs. Tuberculin treatment was begun and the patient improved, only to have subsequent relapses. Recovery was never complete. An excessive dose of tuberculin was administered by mistake by an assistant, and was followed promptly by a most violent focal reaction. This, however, subsided under subsequent small doses of tuberculin.

Studies of the sinuses failed to reveal any evidence of disease. In the summer of 1921 the patient observed an area up and to the temporal side in which he could not perceive light. This area gradually spread past the macula and involved the entire eye. In about three months the eye became blind. In February, 1922,

a similar process invaded the right eye, from above downward, and in a few months light perception was lost. Further studies of the sinuses showed both sphenoids and posterior ethmoids badly diseased. They were opened up and curetted. No recovery in vision was noticeable until about two months ago, when the patient saw light in the lower left-hand field of the right eye. The entire temporal retina is now responsive to light to within 10 degrees of the macula. In the lower left-hand field of the left eye there is light perception and light projection. The apparent hemianopic condition of the restored fields is probably not significant of invasion as far back as the chiasm. The process, in all probability, has been limited to the optic nerves as they pass through the optic foramina, an extension process. *X*-ray examinations of the orbits, including the optic foramina, however, revealed no visible changes in the bone.

The relation of the sinus condition to the tubercular toxins in the production of the eye symptoms is interesting. The fact that recovery was not complete under tuberculin was in all probability due to the diseased condition of the ethmoids and sphenoids. In former articles attention has been called to the necessity of careful study of the posterior accessory sinuses in tuberculous conditions within the eye. In cases which resist treatment by tuberculin and other proper measures it would seem advisable that the sphenoids and ethmoids be most carefully explored and, if necessary, opened up and drained.

Complete Paralysis of the Third and Partial Paralysis of the Fourth Cranial Nerve

Dr. Luther C. Peter presented a patient, aged thirty-two years, who in February last suddenly developed double vision and drooping of the right upper lid, accompanied by a severe pain in the head. She was admitted to the Philadelphia General Hospital and treated with salvarsan and mercurial inunctions. A few days ago she came to the Polyclinic Hospital. At this time she showed a com-

plete ptosis of the right upper lid and complete paralysis of all the muscles supplied by the oculomotor nerve. The eye is in position of extreme rotation. When asked to look down and out there is an abortive attempt at torsion. It is probable that the fourth nerve, while paretic, is not completely paralyzed. A Wassermann test has not been made, but in all probability the palsy is luetic in origin. A surface lesion, as the nerves make their exit from the brain substance, may be responsible for the palsies. The suddenness of the onset and the fact that there were no other complications strengthen the belief that the lesion is either in the nuclear region or in the fascicule before the nerve trunks make their exit from the brain tissue.

Extensive Burn of Face and Eyelids

Dr. Wm. Campbell Posey exhibited a man with an extensive burn of the face, in consequence of which the right lower lid was entirely lost and the left greatly ectropionized. He proposed to correct the deformity by means of a Thiersch graft in the right eye and Esser inlay in the left.

He referred to a case seen by him some years ago of entropion of all four lids, where the cornea had been exposed for twenty years and, notwithstanding this, the cornea had but slightly hazed and vision was practically normal in each eye. He said he preferred pedicle flaps in the restoration of the function of the lids after burns whenever the surrounding skin was healthy. He recalled a case of a young woman whose face was severely burned by vitriol, in consequence of which not only were all the lids ectropionized but the skin of the nose burned away and the mouth much distorted. An attempt at the correction of the ectropionized lids and the covering of the nasal bone by pedicle flaps, all at one sitting, proved futile on account of the difficulty in respiration which arose when the false openings in the nose created by the burns were closed by the flaps. A later operation upon the nose by Dr. John B. Roberts successfully closed these openings.

Dr. Luther C. Peter said that six months ago he saw a similar case in which the ectropion from an acid burn was limited to the upper lids. There was complete eversion of the right upper lid and partial eversion of the left upper lid. The eyelash border was adherent to the skin in the brow and the conjunctiva of the under surface of the lid and the fornix was hypertrophied and redundant. The patient was unable to see because of the deformity and the hypertrophy of the conjunctiva. A small incision was made at the margin of the lid in the brow, the lid dissected loose and turned down into place and sutures introduced into the lid margin and fastened on the cheek by adhesive plaster. Thiersch's grafts were then applied and covered with rubber dam and the eyes bandaged for five days. The patient made a good recovery with lids fairly normal in appearance.

Quantitative Perimetry

Dr. Luther C. Peter called attention to the inadequacy of taking fields with the white test object alone, although this method is probably quite as illuminating as field studies with colors without regard for standards in the matter of light preëxposure, surrounding field and fixation. Without proper standards the Bjerrum method of taking fields on tangent planes naturally grew into favor and with modifications has continued in popularity. Those who have contributed most to the quantitative technic in recent years are Sinclair, Traquair and Walker. That the technic is simple, is not complicated, and is less fatiguing to the patient and the operator is conceded. Its general application, however, to all forms of field studies and its ability to uncover every variety of field change, and the tendency of substituting a quantitative technic for the more accurate qualitative methods, are debatable questions.

Clinical evidence gained, especially in glaucoma, from Bjerrum's sign, Rönne's step, Seidel's sign, etc., has confirmed the correctness of our knowledge of the distribution of the retinal nerve

fibers. Clinical and pathologic studies of the corticovisual centers have also clearly outlined representation in the cortex in a most orderly manner. Macular centers are represented at the posterior poles of the hemispheres and the peripheral retina is serially projected forward along the calcarine fissure. The inferior retina has cortical representation in the inferior lip of the calcarine fissure, while the superior retina is represented in the superior lip. Isolated lesions, therefore, in the retina itself, or in the cortical centers, and even in the optic radiation, may cause only a small peripheral or central defect without involving other fibers. The compactness of the visual fibers of the optic radiations, chiasm and optic nerves, on the other hand, explains the wide extent of the damage when these structures are injured by disease.

The first source of error in quantitative perimetry, therefore, may be in the detection of minute peripheral lesions, because a small test object may narrow the limits of the fields so as to fail to detect a small peripheral cutting, which either the red or blue stimuli would readily pick up. A second source of error will be found in studies of the central field in glaucoma, where the minutest white test object may fail to give evidence of a Bjerrum sign, although a small green test object will readily reveal such a beginning scotoma. Third, in retinal and choroidal disease the behavior of colors cannot be ignored. This is especially true in functional night blindness and in other forms of retinal and choroidal disease.

The distinct field of quantitative perimetry as a part of a good general technic lies first in its use in outlining the dividing line in chiasmal hemianopsias, or even homonymous hemianopsias, when peripheral limits are somewhat contracted. It can be used advantageously after careful qualitative studies have been made to check up any possible increase or contraction of the field. Finally, it serves as a good method for neurologists in making preliminary studies in brain cases. Such studies, however, should be supplemented by good qualitative methods.

Traquair modifies his quantitative method by including colors. The gradual reduction of the colored stimuli is the most valuable

method of picking up minute changes. The fact that clinicians have found color sensitivity a most elusive study, and physicists have devoted much time to the determining of color thresholds, color sensitivity and color limits, confirms our convictions that in pathology we cannot ignore the patient's response to colored stimuli. Quantitative perimetry should therefore take its place as a method in perimetry and should be accorded its real value, but it cannot safely replace qualitative methods without the loss of much useful information in a large percentage of cases.

Tuberculous Iritis

Dr. T. B. Holloway reported the following cases:

Case I.—Colored, male, aged nineteen years. No tuberculous family history. Specific disease was denied. The patient came to the Wills Hospital about January 3, stating that the eyes had been inflamed since March. The globes were moderately injected, each cornea showed rather wedge-shaped infiltrates at the limbus below, with scattered pinhead-sized infiltrations occupying the various layers of the corneæ. In each eye there were numerous synechiæ, but no tubercles were noted in the iris. *X*-rays of the teeth and sinuses were negative; that of the chest had not as yet been received. Nose and throat, urologic and blood-serum examinations were negative. There were vague pulmonary signs, but nothing definite of tuberculosis. While in the hospital for several days the temperature was frequently subnormal.

Case II.—Female, aged nineteen years. First seen at the Wills Hospital in June, 1922. Medical examination revealed a mitral stenosis but no definite signs of pulmonary tuberculosis. Wassermann and urine negative. Teeth and sinuses negative. Keratosis pilaris about eyebrows. *X*-ray of the chest showed adenopathy of both roots, lung picture not diagnostic of tuberculosis. Tension while under observation normal and subnormal. The right eye was moderately injected; there were a few vague

infiltrates in the cornea and moderate iritis, but no true tubercle formation. A drawing, made by means of the corneal microscope, shows widespread iridic involvement with ten to twelve typical tubercles. There was a greater involvement in the lower portion of the cornea than in the fellow eye.

Dr. Wm. Campbell Posey said that he had seen a number of cases of solitary tubercle of the iris, mostly in colored subjects, and had observed the manner in which Descemet's membrane had resisted the intrusion of the tubercular process, notwithstanding the fact that the tubercles were impaled against the posterior surface of the cornea for weeks. Finally, in most of these cases, the substantia propia of the cornea became invaded from the periphery, the bacilli gaining entrance to that membrane through the spaces of Fontana. He had noted in the literature that some pathologists now deny that Descemet's membrane has such protective qualities. In many of his cases the tuberculous process in the eye seemed to be primary, although he understands that it is the general opinion that there must be foci elsewhere in the body. He has found injections of old tuberculin of very great value in combating tuberculous processes in the eye.

Microcornea and Congenital Cataracts

Dr. G. Oram Ring exhibited a girl, aged thirteen years, with the right eye convergent and amblyopic, microcornea and lens cataractous. The left eye, microcornea, a dense, small, white plaque at the posterior lens pole surrounded by an area of less dense opacity with a vision of 20/200.

The mother was married at twenty-five years. First child miscarriage. Second child died at seven months. Third, patient exhibited. Fourth died at five months and had congenital cataracts, microcornea and colobomata of irides. Fifth, girl, living, aged eight years; no ocular defects but undersized. Sixth, miscarriage. Seventh, girl, aged five years, with no eye trouble and fairly healthy.

FEBRUARY 15

Fistula of Cornea

Dr. Luther C. Peter presented a patient, aged sixty-five years, who was operated on for a secondary cataract by discission four years ago. Examination reveals a bleb at the upper outer cornea of the section, about 2½ mm. in diameter, with an elevation of about 2 mm., covered by a thin layer of conjunctiva. Vision with correction, 6/6. In view of the fact that the condition had existed for four years it was thought best not to interfere. The case, however, is presented for the judgment of the members of the section as to the advisability of any further operative procedure.

Cicatricial Ectropion

Dr. George H. Cross described a case of cicatricial ectropion which was exhibited by Dr. Posey at the last meeting of the Section. The deformity resulted from burns in a mine twenty-two months before. There was absence of the lower lid of the right eye and the mucous membrane was continuous with the surface of the cheek, with almost complete obliteration of the lid border. The ectropion of the lower lid of the left eye was not so marked. As the surface of the forehead and face was like parchment, so that a pedicle flap graft or sliding graft could not be secured, epidermal grafts from the arm were taken after the method of Esser and Gillies.

Dr. Posey asked him to operate with him. In the right eye the lower lid was separated from the cheek by blunt dissection through the rather loose fascial attachment and the lid margin separated from the cheek. This restored the lower lid to its original thickness, except for the loss of the surface epithelium. The lid border was sutured to the eyebrow over the upper lid to provide a flat base for the epidermal graft, which was placed over the denuded area. This was then covered with a layer of modeling compound, bandaged and not disturbed for four days, at which time the modeling

compound was cleansed, replaced and removed permanently in about three days. The lid is rather thick on the mucous membrane surface, due to an overgrowth of hyperplastic tissue in the conjunctiva following the long exposure. If massage is carried out on the lid it will flatten out and improve greatly in appearance. Should further rebuilding be necessary, what was done provides an excellent groundwork.

The left eye was corrected by a dissection of the subcutaneous tissue of the lower lid from the skin over an area one-third larger than the surface to be covered, and in this area a proper shaped mold of modeling compound was buried after it was first covered with an epidermal graft taken from the arm, which was wrapped around the mold with the raw surface outward. The skin was then sutured in the line of the original incision, completely covering the buried, skin-covered mold. At the end of five days an incision was made with a sharp knife through the original incision and down through the graft on the mold into the modeling compound. This permitted the edges to retract and the mold to be lifted out, leaving a channel covered with epithelium. In a short time this filled up and the lower lid is now in perfect apposition with the eyeball and the original defect entirely corrected. As in the other eye a course of massage will improve the surface and the skin will be flat and smooth.

Sarcoma of the Ciliary Body

Dr. J. Milton Griscom exhibited a woman, aged twenty-six years, who complained of failing vision in her right eye during the past six months. When the vision was first affected she complained of pain continuing about one week, but subsequently there had been no discomfort. The cornea was clear, anterior chamber shallow down and in, pupil irregularly oval, axis 135 degrees and no reaction to light. The lens, which was slightly hazy, was dislocated down and in so that the upper outer margin could be seen. Back of this a nodular brownish-red mass, adjacent to the ciliary

body and projecting about 4 mm. into the vitreous chamber, was observed. The retina was totally detached. Transillumination revealed a shadow in the area near the insertion of the external rectus. Tension, normal; vision, *nil.*

Dr. Griscom proposed to enucleate the eye and hoped to be able to show the specimen at a later meeting.

Dr. William Zentmayer said an interesting feature of Dr. Griscom's case is the hypotony. While a minus tension is at times seen with an intraocular tumor, it is unusual, especially where we have other conditions usually associated with increased ocular tension, as in this case a partial dislocation of the lens and almost complete obliteration of the anterior chamber. The possible explanation is that the growth has destroyed the function of the ciliary body, thus bringing about a condition of synchisis.

The Treatment of Prolapse of the Iris Following Cataract Extraction

Dr. Howard F. Hansell considered prolapse occurring during operation and prolapse occurring during convalescence.

The substance of his paper is given in the conclusion which he has drawn from his experience: (1) To avoid surgical intervention immediately after the extraction; (2) to allow small stationary prolapse cysts to remain undisturbed; (3) cysts increasing in size, however gradually, should be cut off, the surface of the original incision freshened and brought together by a double-needled suture, the needle introduced from within, outward; (4) the conjunctival flap is practically worthless.

Dr. William Zentmayer concurred in Dr. Hansell's conclusions as to the best methods of treating prolapse, except his final statement that a conjunctival flap is practically worthless. Recently he had had two such cases to treat, following operations by visiting surgeons. In one there was a secondary glaucoma and an attempt to free the iris was unsuccessful.

Dr. S. Lewis Ziegler said that the treatment of iris prolapse is either preventive or remedial. To prevent it one must avoid an irregular or scleral incision, or even a shoulder incision, and make an even, flat bevel of the cornea with a free sweep of the knife held perfectly parallel with the iris. When prolapse occurs the iris should be pressed back by a sweeping movement of the iris spatula. If the vitreous is pressing the iris forward it may be necessary to puncture the posterior capsule and allow some vitreous to escape. A gaping wound calls for corneal suture if the patient is quiet. The conjunctival flap of Kuhnt or Van Lint may also prove useful. The iris must be got out of the wound or else protected by a sheet of beaten gold or thin oiled silk tissue until corneal union has occurred.

Dr. Thomas H. Fenton reported the appearance of the prolapse of the iris following cataract extraction in 6 cases, two to six days after operating, due to insubordinate and refractory behavior of the patients. Their recovery was uneventful although operative relief had not been undertaken.

Dr. George H. Cross stated that he would like to say a few words in defence of the conjunctival flap: As Dr. Hansell had been so emphatic in his statement that the conjunctival flap proved of no value in prolapse of the iris after cataract extraction, he felt his remarks might be misinterpreted to include the use of the flap in other procedures than the one under discussion. He felt that in certain forms of injuries the conjunctival flap had been of most valuable assistance.

Dr. Hansell stated, in conclusion, that he may not have expressed himself clearly. The remark that the conjunctival flap was practically worthless is applicable only to the subject under discussion. The conjunctival flap in punctured wounds and other recent injuries to hasten healing is of the greatest value and has been practised by him with entire satisfaction for many years.

Slit Lamp Studies of Hernia of the Vitreous and its Relation of Cataract Operations: A Preliminary Report

Dr. Luther C. Peter reported several cases of hernia of the vitreous into the anterior chamber: (1) after spontaneous absorption of traumatic cataract; (2) after discission of the membrane cataract; (3) after extraction of the lens by the Barraquer, Smith and the ordinary method with capsulotomy. He called attention to the advantages of studying the anterior chamber, the lens and the anterior vitreous body by means of the slit lamp and corneal microscope with low magnification (24 times).

In the traumatic group were 2 cases of spontaneous absorption in which prolapse of the vitreous was present.

Four cases of discission for after-cataract showed in each instance vitreous hernia. All the cases of this group probably are followed by vitreous prolapse.

Two Barraquer operations were examined. Externally conditions seemed to be perfect, excepting for tremulous irides. Internal examination, however, revealed free vitreous in the anterior chamber and marked vitreous opacities. Two Smith operations were examined. One performed by Smith presented incarceration of the iris, a wide coloboma and a drawing up of the iris into the wound. Slit lamp studies showed the vitreous held well in place by a firm and smooth hyaloid membrane. There were no opacities. The second Smith case was operated on by the author. The coloboma was unduly wide but the iris was not drawn up. The vitreous was held well in place. A few opacities were present.

Extractions performed by the usual method with capsulotomy did not show any tendency to vitreous prolapse.

Although the number of cases was too small from which to draw general conclusions, the cases of Barraquer and Smith, when compared, tend to indicate that more trauma is exerted on the delicate hyaloid membrane by the suction method than by that of Smith. Vitreous opacities were also marked after the Barraquer extractions. In every case of vitreous prolapse in all groups there was

iridodonesis. This latter condition was absent in those cases in which there was no prolapse of the vitreous.

He pointed out that the dangers in the anterior chamber, such as secondary glaucoma and detachment of the retina, are the most serious of the complications.

The author suggested that careful examination of the interior of the eye after the various cataract operations would tend to a greater respect for the integrity of the vitreous and the hyaloid membrane and would encourage operators to perfect their technic in cataract extractions so as not to necessitate a discission for after-cataract. A further report will be made by the author after a larger number of cases have been studied.

Dr. S. Lewis Ziegler said that study of the intraocular tissues with the slit lamp was fascinating. As to the vitreous transfer from one chamber to the other he thought there were limitations. He had always practised his one V-shaped operation, which so supported the vitreous body as to prevent its migration. He preferred to call this dislocation of the vitreous where it followed looping of the lens or the Smith-Indian extraction. Here the whole body came forward, pressing the iris into a wide iridectomy and permitting a tremulous iris.

In the other case of sclerocorneal fistula with vitreous exposure following a too free discission he would suggest reinforcement by a Van Lint conjunctival flap or else a mild cauterization to cause interstitial thickening of the conjunctiva and thus remove the danger of future infection from an intercurrent attack of conjunctivitis.

Dr. Peter, in conclusion, said the slit lamp, although of much scientific value, can only be of real value in general clinical work in so far as it can be made to contribute to the refinement of clinical diagnosis.

If Dr. Ziegler referred, in his operation, to an incision through and through the lens, it is practically impossible to escape incising the hyaloid membrane. The hyaloid membrane and the posterior capsule are necessarily in contact. A discission operation which passes through the posterior capsule must also go through the

hyaloid membrane and a vitreous hernia will naturally follow. The extent of the hernia will depend largely upon the consistency of the vitreous body.

He regretted that he could not agree with Dr. Holloway in reference to the relative amount of trauma in the Barraquer and Smith operations. We are not, in this discussion, comparing the operative ability of the two advocates of these methods, but the two operations in themselves. The Smith operation, he feels sure, can be performed with a minimum amount of trauma and with excellent results. The Barraquer operation, on the other hand, has a twofold source of trauma: first, the hard, non-resisting instrument which is introduced into the anterior chamber is too inelastic to accomplish its work without damage to the hyaloid membrane even in expert hands, and in the second place the vibrations of the suction apparatus are probably a large contributing factor in the intraocular trauma resulting from the operation.

A Case of Mikulicz's Disease

Dr. McCluney Radcliffe presented a case of Mikulicz's disease in a colored man, aged twenty-four years, who was admitted to his clinic at the Wills Hospital during January, 1923.

In December, 1922, the patient first noticed swelling of the upper lids, which were not tender to touch, following a diseased tooth. Examination showed bilateral enlargements of the lacrimal, parotid and submaxillary glands. The accessory parotid and submaxillary glands were not enlarged. The preauricular nodes showed bilateral enlargement. The cervical chain of glands showed a questionable enlargement. The tonsils were evidently enlarged, as they had been removed four months previously, but did not influence the lymphomata. He had several infected teeth and also an involvement of both ethmoid regions, as shown by the skiagraphic examination. The Wassermann and von Pirquet tests were negative.

The treatment suggested was drainage of the ethmoids, *x*-ray, thyroid, arsenic and the iodides.

Dr. William Zentmayer said that it might be of interest to review von Mikulicz's views of this disease today. In a recent edition of his book, *Diseases of the Mouth,* he says there is no genesis for it. This has been shown by von Brunn, who states that the cases reported exhibit a continuous chain of slight variation from those which show a symmetrical hypertrophy of the lacrimal and salivary glands to those in which the spleen and other lymphatic glands are involved (pseudoleukemia) and cases in which there are also associated blood changes (leukemia). The histologic changes in the glands also speak for a varied pathogenesis, as in some cases they present lymphocytes with eosinophiles and in others inflammatory and cicatricial changes. In other words, there is a lymphomatosis of unknown origin, probably in most cases, however, toxic. He points out that such a lymphatic enlargement may be produced by certain agents such as iodin or lead. Endocrine origin has been suggested, and 1 case is recorded where this affection was associated with congenital disease of the pituitary body.

Dr. S. Lewis Ziegler said that the above was a typical example of Mikulicz's disease. He had reported 3 cases and seen 2 others. The chief factors of interest were etiology and treatment. In his cases respiratory obstruction, either tonsillar or nasal, was the sole cause. No one had isolated any microörganism from the excised glands. The hyperplasia is a true lymphoma caused by the irritation of perverted chemistry in the affected glands. The sympathetic system is at fault. The curious recession during an acute intercurrent disease and recurrence after this is cured show this. Osler's case disappeared during pleurisy but recurred after convalescence. Similar experiences following pneumonia, appendicitis, cholera and erysipelas confirm this observation.

As to treatment, whatever increases oxidation brings relief. Tonsillectomy and nasal cauterization cured his cases through restored breathing. Dr. Radcliffe kindly asked him to examine his case, which showed respiratory obstruction from polypoid degeneration of the middle tubinate and excessive purulent secretion from the sinuses. He would suggest, first, limiting treatment

to the nose and watching the result. After that, arsenic, the iodides, pilocarpin or apomorphia in small doses (gr. 1/200) at frequent intervals will increase lymphatic action and stimulate oxidation. The *x*-ray reduces the lymphoma but destroys the parenchyma of the gland.

MARCH 15

An Unusual Corneal Lesion

Dr. Edwin B. Miller exhibited a woman, aged sixty-one years, who presented herself July 1, 1918, complaining of bad vision in the right eye. Examination O. D. showed an old adherent circular leukoma, 2 mm. in diameter, just inside the limbus at 6 o'clock. Vision: R., 1/30; L., 5/30. Fundus examination revealed many flame-shaped hemorrhages in and about the macular region. Under treatment this cleared up and, with correction, V = 5/6 in each eye.

On December 10, 1920, vision of the right eye was suddenly reduced to counting fingers at one foot, because of posterior uveitis with opacities. Under treatment vision improved.

On February 19, 1923, examination of the right eye showed no change. Left eye, vision, = 5/9. Just inside of the limbus, between 2 and 3 o'clock, there was a circular area, 4 mm. in diameter, slightly thickened on the margins and thinner in the center, vacular, and with a few areas of white infiltrate inside the circular area. From the opposite margin to the center of the cornea there was a very faint striated areola, about 2 mm. wide. One week later there was a still fainter, gray, striated areola reaching about 2 mm. beyond the first to the center of the cornea. These striated areas have nearly coalesced, but there is still a faint line of demarcation. The whole lesion is beneath the anterior epithelial layer of the cornea. Von Pirquet is positive. Wassermann is negative. Urinary analysis shows a trace of albumin and a few hyaline and an occasional granular cast.

Dr. Miller said he had not been able to determine the diagnosis, although a non-typical form of disciform keratitis might be considered.

Dr. William Zentmayer said that, while he had had no experience of his own, he recalled that some years ago Dr. Randolph presented a case which resembled Dr. Miller's and represented probably an earlier stage of the same condition. There was some question as to the diagnosis, but the general opinion was that it was a hyaline degeneration. A year or two later Dr. Posey exhibited before the Section a case showing almost exactly the same condition as in Dr. Randolph's patient.

Dr. Wm. Campbell Posey advised cauterization by desiccation.

Some Observations of Alopecia and Poliosis of the Lids and Brow, with Report of Cases

Dr. Wm. Campbell Posey, under the above heading, cited an instance of a young married woman, with moderate anemia, who lost all the eyelashes from the left upper lid in about four months' time. The hair of the rest of the body was unaffected. Cases similar to the foregoing, illustrative of the condition designated as alopecia of the eyelashes, by which is understood the partial or entire absence of the cilia due to conditions other than local inflammations of the lid, are rare. The literature contains quite a few instances of congenital origin. Dermatologists record the conditions as occurring in leprosy, leukoderma, pityriasis and seborrhea. Syphilis is a frequent cause of alopecia, gumma of the lid depriving that structure of its lashes in a complete and characteristic manner, while the cilia may fall out without previous inflammatory symptoms, in association with the loss of hair in other parts of the body, which is so frequently observed in constitutional lues. In 136 cases observed in the early stages of syphilis, Wilbrand and Saenger found a loss of cilia alone in 5.1 per cent; of brow alone in 8.8 per cent and a simultaneous disappearance of cilia and brows in 33.3 per cent. J. T. Shoemaker

reported an interesting case following an attack of measles, and thought it probable that the thyroid and gonads are at fault in many cases of alopecia. A number of authors observed the condition in association with Graves' disease. Michel asserts that the process is in all probability a trophoneurosis. The case reported by the writer is probably of this origin, doubtless associated with some obscure nutritional change, emanating from disordered glandular function. Hysterical girls and the insane not infrequently pull out the lashes and sometimes the hair of the brow and head.

Closely allied to alopecia is poliosis of the lids, a loss of color and graying and whitening of the lashes. This condition, which is not nearly so rare as the former, Dr. Posey illustrated by reporting an instance where the hair of the right eye brow and the lashes of the corresponding side of the face became blanched during a period of three months without known cause. Despite tonic treatment, etc., the condition persisted. Poliosis may be congenital.

In accounting for the discoloration of the lashes, Dr. Posey quoted Stellwagon's opinion that the rapid evolution of air vesicles in the hair follicles was probably the sublying factor. Although very much more rare than one would be led to believe by the frequent accounts in the general literature of the hair becoming white overnight from grief or shock, Dr. Posey cited well-authenticated cases in medical literature where such had been the case. He gave the notes of a number of cases where the cilia became discolored from repeated attacks of migraine and also in association with painful affections of the eye-ball, especially in sympathetic ophthalmia. He quoted Vogt's figures showing a connection between poliosis and some form of iridocyclitis, especially that due to sympathetic disease, as occurring in 39 per cent of all cases in the literature. A number of instances have been reported as occurring in perfectly healthy individuals. In one instance the discolorations came on overnight; in others in a few days; while in others the period varied from five to fifteen days to several months.

Dr. Jay F. Schamberg said that in the presentation by Dr. Posey

there were two aspects of the subject which had dermatological bearing: The loss of hair, or alopecia; and the whitening of the hair, or poliosis—a condition more commonly referred to in dermatological literature as "canities."

Alopecia in patches may be due to two different types of causes. It may be due to an interference with the nerve innervation of the hair or it may be of microbic or of parasitic origin. There are in the literature authenticated reports of epidemics of spotted baldness occurring upon the scalp which have been reported both in this country and more particularly in Europe. Bowen and Putman, in Boston, have reported outbreaks of this condition in an institution for young girls, in one of which 63 out of the 69 girls in the asylum developed the affection. This is, of course, convincing evidence of the possible infectious nature of this condition. But such epidemics are rare and unusual, and the ordinary type of alopecia areata as we see it in practice is of nerve origin. The condition may follow shock, fright, grief and similar causes. As experimental proof of the nerve origin of this condition, it may be said that Joseph, in 1886, produced alopecia areata in cats by section of the cervical sympathetic nerve, and later two investigators caused the bald areas in other animals by excision of the second cervical ganglion. It would appear likewise that some focal infections in teeth and tonsils and reflex irritation from impacted molars may in some cases be responsible.

The whitening of hairs is likewise a condition due to nerve influences. There was at one time considerable skepticism concerning the observation that the hair might "turn white in a single night," but in recent years there has been ample confirmation of this observation. A number of individuals who were under great mental stress in the recent war were observed to have their hair turn gray or white within a very few days. In my own practice I saw a woman who was the eye witness of the accidental electrocution of her only child, who informed me, and the statement was corroborated by her husband, that her hair had turned completely white within two or three days following this calamity. The loss of color is alleged to be due to an infiltration of the hair with air.

Whether this is actually the explanation or not is not definitely known. It would be difficult to apprehend how the entire pigment could be absorbed or lost within so short a period of time.

Dr. Burton Chance said he desired to place on record 3 cases which fell under the classes presented by Dr. Posey—2 of alopecia and 1 of poliosis. Those of dropping out of the lashes and brows were, first, a lady, in her thirties, who when she was a young girl became affected after an attempted assault upon her by a ruffian. The other, the daughter of a physician and the wife of a physician, who had lost lashes, brows and scalp hair by the end of the week after the difficult and complicated labor with her first child. Nothing in the years which had elapsed had been of avail in either case to restore the lost hairs.

The case of poliosis was seen in a young robust lady whose black hair, brows and lashes were changed to gray overnight, several years previously, after witnessing the tragic accidental death of her father.

The Use of the Prism Scale to Neutralize and Center Spectacle Lenses

Dr. S. Lewis Ziegler called attention to the fact that his double prism scale, in black and red, which he first presented before the American Medical Association about thirty years ago, had other uses than the measuring of prismatic strength. He had found it most useful as a daily help in neutralizing and centering spectacle lenses, and especially in testing bifocal segments for relative vertical decentration, which, if undetected, is so liable to produce an artificial hyperphoria for the patient.

He gave a minute description of the method of using the prism scale, and showed illustrations of prisms set with base horizontal, base vertical and base at an angle, otherwise known as resultant prisms. He also called attention to his table of equivalent and resultant prisms, in which the prisms to be ordered were recorded in whole numbers, always remembering the dictum that "the

vertical prism must be exact but the horizontal prism may be approximate."

His conclusions were that: "If the superimposed lenses are held evenly before the junction of the two scales, and at exactly one meter's distance, it can at once be seen (1) whether the lens is properly neutralized, (2) whether it is accurately centered and (3) whether the prismatic strength is correctly measured."

On the Local Control of the Ocular Circulation

Dr. Francis H. Adler stated that the dependence of the intraocular pressure on the general blood-pressure has long been known, and the effects of changes in blood-pressure has been the subject of many investigations. Intraocular pressure follows qualitative changes in the blood-pressure, but not quantitative always. This and many other facts have led to the assumption that there is a mechanism in the eye whereby the local pressure in the intraocular bloodvessels could vary independently of the general blood-pressure. It may be assumed that this local control of the ocular circulation is brought about by vasomotor nerves to the bloodvessels, and many observers have attempted to prove the existence of such nerves.

In 1904 Henderson and Starling gave a critical review of the literature, and by a series of well-controlled experiments proved that stimulation of the head-end of the cut cervical sympathetic caused a rise in intraocular pressure on the side stimulated. This rise was of sudden onset and short duration, and was followed by a gradual and more or less persistent fall below normal. The general blood-pressure remained constant through the experiment. They proved that the initial rise was due to the contraction of unstriated muscle fibers surrounding the globe in Tenon's capsule, which fibers are supplied by the sympathetic. The fall in pressure they attributed to a constriction of the intraocular bloodvessels, through the agency of vasoconstrictor fibers in the sympathetic.

In 1912 Hill and Flack repeated these experiments, and although they obtained identical results they were unwilling to

draw the same conclusions from them on the ground that uncontrolled factors other than a vasoconstriction might give rise to a fall in intraocular pressure. We would point out, further, that the fall in pressure in Henderson and Starling's experiments might be explained on the following basis: The compression of the globe caused by the contraction of the unstriated muscle in Tenon's capsule by raising the pressure might squeeze fluid out through the normal avenues of escape. When the compression ceases there would be a diminished volume of fluid in the eye which would reveal itself in lowered intraocular pressure, persisting until the amount of fluid lost was made up from the blood stream. We see this clinically as the effect of massaging the eye-ball, simple pressure on the globe and repeated application of a tonometer. It seemed advisable then to attempt a series of experiments which would show conclusively the direct effects on the ocular blood-vessels of stimulating the cervical sympathetic. For a full description of the method the original article will have to be consulted. It consists essentially in measuring the rate of formation of fluid in the eye at normal intraocular pressure, but with raised blood-pressure. When a constant rate of fluid formation has been brought about the sympathetic is stimulated and the effects on the rate noted. If the blood-pressure remains constant throughout, in the absence of other factors, the rate of fluid formation must depend on the size of the intraocular bloodvessels.

The experiments were divided into two sets: (1). Those in which Tenon's capsule was intact. (2). Those in which Tenon's capsule has been cut completely through around the globe, so as to destroy any effect of its unstriated muscle fibers.

In both cases we found that stimulating the sympathetic caused a marked decrease in the rate of formation of fluid, which only gradually returned to normal. When Tenon's capsule was intact there occurred during the first few seconds of stimulation a rapid increase in the number of drops formed, which was proved to be due to the contraction of the unstriated muscle fibers, as described by Henderson and Starling. The blood-pressure remained constant throughout.

CONCLUSIONS. 1. A rise of blood-pressure causes an increased formation of aqueous humor even when the intraocular pressure is maintained at its previous normal level.

2. The rate of formation of this fluid is diminished by stimulating the cervical sympathetic, a fact which can only be adequately explained by constriction of the intraocular bloodvessels. Whether this vasoconstriction is a factor in preventing sudden changes in blood-pressure from affecting intraocular pressure can only be determined by further experiment.

3. The effects on the unstriated muscle in Tenon's capsule, as explained by Henderson and Starling, are confirmed.

UNILATERAL EXOPHTHALMOS: A CLINICAL REPORT OF FIVE CASES

(By Invitation.)

Dr. Karl Musser Houser reported 5 cases of unilateral exophthalmos studied and treated in the neurosurgical clinic of Dr. C. H. Frazier at the University Hospital. In each of the reported cases different etiologic factors gave rise to the condition. In one the intracranial lesion gave rise to a train of symptoms of such nature that a diagnosis of orbital tumor was made. Another demonstrated orbital abscess and the difficulty that may be experienced in correctly diagnosing the condition. A third case illustrated the proptosis that may appear where total ophthalmoplegia exists. Still another case demonstrated cavernous sinus thrombosis where extensive operative interference failed to give relief. A fifth case dealt with pulsating exophthalmos due to arteriovenous aneurysm where subjective symptoms were relieved by carotid ligation.

Dr. Wm. Campbell Posey said he spoke from personal experience of Dr. Frazier's admirable technic in operating upon the orbit. Like him, he had found the Kronlein procedure of great

value, particularly since its modification by Magitot. Mucocele of the accessory sinuses of the nose had occasioned the majority of cases of unilateral exophthalmos seen by him, those of the ethmoid and frontal cells predominating. Graves' disease is an occasional cause of unilateral exophthalmos. *X*-ray is of great value in the diagnosis of orbital conditions, particularly in those complicating sinus conditions.

Dr. H. Maxwell Langdon said he desired to report the case of a colored man, aged thirty-nine years, who visited the clinic of Dr. Charles W. Burr at the Orthopædic Hospital on Monday, March 5, 1923, suffering from exophthalmos bilateral and with loss of vision of the left eye, associated with impaired usefulness of the left arm owing to stiffness at the shoulder.

Examination showed considerable rigidity of the left shoulder with much wasting of the deltoid muscles, making it impossible to lift the arm above the head.

The right eye was found to have normal vision and to be normal in all particulars. There was a complete ptosis of the left upper eyelid.

Pupil dilated and fixed and complete loss of ocular rotation, except a very slight intorsion resulting from a slight action of the superior oblique. Vision was lowered to faint light perception. Ophthalmoscope revealed media clear, disk normal in color and outline, with slight fulness of both the retinal arteries and veins. Hertel's exophthalmometer showed a proptosis of 13 mm. on the right side and 15 mm. on the left.

The cause for the disability in the shoulder was found on *x*-ray examination to be due to an old fracture. The loss of vision and loss of motility of the eye suggest some inflammation of the tissues back of the orbit. As the other eye was unaffected it must be in front of the chiasm, and since the structures coming through the optic foramen and sphenoidal fissure are all involved in the process it seemed most likely to be orbital. A Wassermann reaction of 4 plus suggested a gummatous involvement of the orbital tissues.

A Case of Steel Imbedded in Posterior Sclera Without Visual Changes

Dr. H. Maxwell Langdon reported the case of a man, aged twenty-three years, who reported at the Presbyterian Hospital on January 30, 1923, with the history that he had been struck in the left eye by a small steel chipping while working in a machine shop.

Examination showed right eye normal. A slight conjunctival hemorrhage to the nasal side of the cornea about 7 mm. from the limbus was noted in the left eye. Slightly down and in could be seen the wound of entrance. Ophthalmoscopic examination showed some slight vitreous haze mostly localized down and in near the wound, while down and in from the disk was a patch of hemorrhage and exudate about three disk diameters from the disk, about 3 x 6 mm. in size. Vision, 5/5.

The question to be decided was whether the steel had passed clear through the globe into the orbit, had become embedded in the posterior portion of the sclera, or whether it had possibly struck the posterior portion of the globe and rebounded to be caught in the exudate in the anterior portion. It is quite obvious that treatment for the first two conditions would be very different from that for the last.

X-ray localization placed it in the sclera at the site of the posterior disturbance, and it was decided not to interfere with it. The hemorrhage in the vitreous completely absorbed and nothing is left but a small patch of atrophy at the site of the original injury.

Dr. George H. Cross stated that Dr. Langdon's cases recalled that of a man with a foreign body just inside the sclera below the ciliary region, localized by Dr. Sweet. He decided to let the foreign body remain, and the man had had no trouble during the past four or five years. His vision remained normal.

Two weeks ago he had a case of foreign body in the vitreous, which had passed through the sclera, about 8 mm. to the nasal side of the limbus. With the ophthalmoscope it was possible to see the foreign body and numerous hemorrhages on the retinal

surface. As it was a chip from the head of a chisel and not embedded, he removed the foreign body. Vision, 20/30.

A third case of foreign body seen by him recently showed by *x*-ray a very small flake-like body which had penetrated through the lens stroma. It was impossible to draw this foreign body into the anterior chamber with a magnet. Examination revealed a small opacity on the anterior capsule of the lens and on the posterior surface a large reticulated exudate similar to a snow crystal. This was gradually absorbing. This case and the one he presented to the College two months ago, where there was penetration of the anterior capsule without cataract formation, caused him to wonder whether it is the rupture of the anterior capsule or the rupture of the posterior capsule which precedes the formation of a traumatic cataract.

Dr. William M. Sweet said he had seen a number of cases similar to the one exhibited in which there was an area of choroidal pigmentation to indicate the point where a small foreign body had penetrated the retina and choroid to become imbedded in the sclera or pass through into the orbit. He recalled only 1 case in which a small piece of steel could be plainly seen imbedded in the retina, where it had remained for a number of years without causing any inflammatory symptoms or reduction in vision. The case was seen in the early days of the use of the *x*-ray, when an exposure of from six to eight minutes were required to secure a shadow of foreign body in the eye. Several exposures were made, but either because of the smallness of the body or the inability of the patient to hold the head and eye steady for the time of the prolonged exposure to the rays no shadow could be secured of the fragment.

Dr. William Zentmayer remarked that Dr. Sweet casually mentioned a subconjunctival hemorrhage in one of his cases. While he had had no large experience in double perforations of the eyeball from foreign bodies, he had twice found subconjunctival hemorrhage as a symptom, and while he did not believe that its absence was of any value in differential diagnosis, yet its presence should lead us to suspect a double perforation.

A Case of Juvenile Cataract in a Child of Eight

Dr. H. Maxwell Langdon reported a case of a boy, aged seven years, who was first seen by him November 3, 1921, suffering from juvenile cataract. History of the patient showed that two years before he had been examined by an oculist and given glasses to correct a beginning convergence of the left eye. At that time no evidence of lenticular changes was noted.

Examination showed no external changes. O. D. V. 5/10 corrected to 5/6; O. S. V. 5/15 corrected to 5/6 plus. Both lenses showed striæ down and in, especially the left eye, which seemed to be more in the posterior cortex. The media were otherwise clear and no fundus changes observed. He was referred to Dr. Ralph Pemberton, at the Presbyterian Hospital, for metabolic studies and a general physical examination. A very complete examination was made of the blood chemistry, urine and in fact the child's whole make-up. Nothing was found wrong except a rather high blood sugar, which was 150 mm. to 100 cm. of blood, and very bad, infected tonsils. The tonsils were removed, and, while there had been some progress in the lens change from November 3, when first seen, and December 22, since February 9, 1922, there has been no advance whatsoever. Vision of the right eye remained 5/5 partly, left eye 5/7.5. Dr. Pemberton has found, and this has been confirmed by the other workers, that a focal infection such as infected tonsils, apical abscesses of the teeth and so on will produce a departure from the normal of the blood chemistry for carbohydrates—in other words, an increase in the blood sugar and a lowering of the tolerance for sugar—and when this focus of infection is removed the blood sugar chemistry will return to normal. Burge has found that lenses with an excessive sugar exposed to light will become cataractous.

The cessation of progress of the lens changes suggests that possibly the infected tonsils by lowering the ability to assimilate starches may have been the cause.

1. A Squint Case Operated on by the Recession Method. 2. A Case of Anophoria

Dr. Warren S. Reese exhibited, first, a squint case operated on by the recession method; second, a case of anophoria.

Case I.—W. C. had a convergent squint of 40 degrees, the left eye squinting. A recession operation was done on the left internus, at the completion of which the visual axes were nearly parallel. They were the same four days later. The bandages were then removed because of a conjunctivitis, and when next seen there was convergence of 10 degrees or 15 degrees. Ten days after operation the squint was measured and there was residual convergence of 18 degrees—in other words, the recession operation had taken care of 22 degrees of the squint. A Reese resection of the left externus was done and the patient's visual axes are now (two months after operation) parallel, the corneal images being exactly symmetrical for distance.

The recession operation is as follows. The left internus was exposed and advancement forceps applied, and the tendon severed as in an advancement. The muscle and tendon were then freed from all attachments to Tenon's capsule. One end of a double-armed suture was then passed through the superficial layers of the sclera, about 6 mm. or 7 mm. back of the original insertion, the needle being carried through parallel to the limbus. Both ends of this suture were then brought out through the end of the severed tendon, then through the conjunctiva, over which the suture was tied. The conjunctival opening was then brought together with two interrupted sutures.

In this case there is slight limitation of adduction of the operated eye, but no sinking of the caruncle such as is seen after a tenotomy.

Case II.—H. J. A. Both eyes are up under cover, the movement in the right, however, being more of a clockwise rotary motion when uncovered. With the Maddox rod over the right

eye there is right hyperphoria and with the Maddox rod over the left eye there is left hyperphoria. Although the left eye is more markedly up under cover the right hyperphoria is greater than the left hyperphoria.

Stevens states that this condition results from the type of cranium. Thus in the mesocephalic type, and especially if the line of the face is that of orthagnathism, the rotations are so generally high that, as a rule, there is anophoria. He also says that anophoria does not indicate a weakness or overaction of any sort of muscles but is an adjustment coincident to the type of head. Later, however, he says that he is convinced that in explanation of the phenomenon of each eye deviating we are to look to the declination, for when by tenotomy the upward rotation of anotrophic eyes has been reduced to the standard of 33 the eyes will still squint upward in exclusion. On the other hand, if the extreme declination is materially reduced the upward deviation ceases. We may regard the deviation as synergic. With the adjustment of the fixing eye to correct an extreme declination the forcible action of the superior oblique may be demanded. With this strong action the fixing eye is depressed, but to counteract this depression in order to effect fixation at the horizon the superior rectus comes strongly into action. Simultaneously and synergically with this the superior rectus of the excluded eye acts, but since there is no occasion for the depressing action of the oblique that eye is raised.

This later explanation seems very plausible. In the few cases of anophoria the writer has seen there has always been a torsion movement of one or both eyes, indicating that the oblique muscles were in some way responsible or involved in this anomaly.

Exhibition of Three Cases of Family Cataract

Dr. McCluney Radcliffe exhibited three little girls, aged five, eight and ten years, with hereditary congenital cataracts in both eyes. The line of heredity was on the mother's side, as the grandmother, twin sister of mother and her brother also had cataracts.

The interesting feature in the cases was the excellent results following the "through-and-through," or Ziegler operation. The operation consists in making a V-shaped incision through the entire lens, including the posterior capsule. There was no reaction following the operation except one eye, and that yielded rapidly to appropriate treatment. He strongly advocated the operation as a safe and efficient surgical procedure.

Dr. S. Lewis Ziegler stated that Dr. Radcliffe should be congratulated on his results in six complete V-shaped discissions of congenital cataracts. It is evident that the slow solution of the lenses was due to the presence of calcareous material in the cortex. He was glad to know that Dr. Radcliffe could verify his statement that through-and-through incision of the lens is practically free from reaction. The one eye that has been slow in dissolving shows evidence of closure of the cystic wound, which has caused localized pocketing or encapsulation of the cortex. He will either have to repeat the discission or perform a paracentesis, as the cortical matter will not dissolve without some encouragement. In all the other eyes the pupillary area seems to be quite clear of lens cortex.

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A Case of Plastic Operation on the Eyelids Following Sulphuric Acid Burn

Dr. Luther C. Peter exhibited a patient, aged thirty-three years, who had a severe burn of the upper part of the face from nitric and sulphuric acid in January, 1922. At the time of the first examination, April 6, 1922, the patient presented scar-tissue formation extending from the right temporal region through the eyelids to the left temporal region. The eyelids were completely everted and the lid borders firmly attached to the skin in the eyebrows. There was complete eversion of the right lid with hypertrophy of the conjunctiva, which completely closed the right

eye. The redundance in the left eye was not quite so marked and the patient was able to see with his head thrown back.

The surgical procedure practised consisted, first, in following the line of cleavage by a blunt dissection; the lids were turned down into place and held taut by sutures, which were fastened by means of adhesive to the lower part of the cheek; Thiersch grafts were then applied; the lids were covered with rubber dam and firmly but evenly packed with cotton, and the eyes were closed by a firm, occlusive bandage. After recovery the redundancy of the tissue in the right eye continued for three or four months. This was treated by tri-weekly applications of tannate of glycerin and by massage. The patient has made a complete recovery. The redundancy in the conjunctival tissue has completely disappeared.

A Case of Plastic Operation of the Orbit Following an Exenteration for Sarcoma of the Orbit

Dr. Luther C. Peter also showed a patient, aged six years, who was presented before the Section about three months ago by Dr. Miller. He had suffered from a sarcoma of the orbit of rapid growth, from six to eight weeks, for which a complete exenteration of the orbit had been performed by Dr. Wm. Clark by fulguration. There was considerable destruction of the lids. Some weeks following the exenteration the tarsi were disengaged from each lid, the eyelash border was removed and the lids were turned into the orbit. The orbit now is completely covered by a good skin lining, with the exception of a small opening at the apex, with which the ethmoid cells communicate. Although the operation was performed in June, 1922, there has been no evidence either of recurrence or of metastasis. This is probably due to the fact that the exenteration was performed by fulguration; that 60 mg. of radium were introduced into the orbit for twelve hours and that subsequently the x-ray was applied. The condition which confronts us now is the closing of the sinus which communicates with the ethmoid cells. It is for this reason that the child is brought

before the Section again tonight. The operation which he proposed to do was to turn a pedicled flap of the thickness of the cheek from the cheek, where the blood supply is good, into the denuded area. The case is brought before the Section for suggestions as to whether this method will offer the best means of closing the part that still remains denuded.

Sir William Bowman and the Bowman Lecture

Dr. Burton Chance read a memoir of Sir William Bowman, who was born in 1816 and died in 1892; his life, therefore, spanned the period of the rise and development of scientific medicine as expressed in the nineteenth century. Leaving school at sixteen, Bowman was apprenticed for five years to a surgeon of Birmingham. At twenty-three he was already exhibiting the products of his studies in anatomy and physiology, as shown in his descriptions of the histology of the kidney, unstriped muscle and the mucous membrane of the intestinal tract, which he illustrated by quite marvelous drawings, which works gained for him the Fellowship of the Royal Society at the early age of twenty-five. At thirty-five, notwithstanding the fact that he was the foremost anatomist and surgeon of England, he departed from that course and devoted himself to ophthalmology, in which field of scientific and practical effort he became the greatest of England and Europe. His operative dexterity was the marvel of his colleagues. His coöperation with von Graefe and with Donders is one of the conspicuous events in the history of ophthalmology. In his time he advanced microscopy, practical surgery and ophthalmology, while his contributions to the anatomy and the surgery of the eye gave him a supreme place in the study of the functions and the diseases of that organ. In his personality he exhibited the greatest kindliness and courtesy coupled with the capacity for arduous study and devotion to business. His benefactions were numberless, chief among which, of special interest to us, were his association with the Ophthalmological Society of the United Kingdom and his

coöperation with Florence Nightingale in the establishment of methods for the training of nurses.

The "Bowman Lecture," to set forth from year to year the advances made in ophthalmology, was founded in his honor, and he lived long enough to welcome several of the lecturers. The memoir was read to signalize the invitation extended by the Ophthalmological Society to that distinguished Fellow of the College of Physicians, Dr. de Schweinitz, who goes this spring as the first representative from America to deliver the Lecture.

A Case of Chronic Simple Glaucoma

Dr. J. Monroe Thorington exhibited a case of chronic simple glaucoma in a colored man, aged thirty-six years. He first noticed four months ago that he was seeing "rings around the sun," and that the left eye was practically blind. On reporting for treatment, O. S., vision limited to light perception; O. D., VI/VI, with field contracted to 7 degrees. O. D. T. = 40; O. S. = 55. No pain at any time. Under eserin in ten days the tension of O. D. decreased to 30, O. S. remaining unaltered. The cupping in each disk is about 5.00 D.

Dr. Ziegler said that the accepted dictum in cases of chronic simple glaucoma has been that "operation is disastrous to vision, therefore, stick to eserin or pilocarpin routinely." But the patient is not always conscientious in using myotics, and sooner or later the case passes from chronic simple glaucoma over to the congestive type, with a sequela of sudden blindness unless immediately relieved by operation. He thought that von Graefe's iridectomy had been more fatal to the vision of such eyes than Elliot's trephining, but his own preference in these cases is a resort to double posterior sclerotomy.

He had recently operated on two cases of this type. The first case had been under his personal supervision for more than ten years, showing only slight changes in field and vision, but with occasional attacks of obscured vision. There were frequent changes

in refraction and heterophoria. She was recently stricken with severe postpuerperal nephritis and placed under the care of another. When again examined, O. S. showed acute hemorrhagic glaucoma, T + 3; pupil widely dilated; vision *nil.* O. D., acute congestive glaucoma, T + 2; pupil dilated; vision, 20/50 J–6. Slight changes in the lens made it evident that cataract would follow an anterior operation. He had therefore advised double posterior sclerotomy O^2. Incision was made with a broad Graefe knife, down and out and down and in, the blade being turned at right angles, after Parinaud, and leaned backward, so that the inner choroidal incision would be a trifle larger than the external. Pain and trembling indicated the possible advent of a choroidal hemorrhage, which was wholly averted by the prompt injection of pituitrin into the arm. The tension in O^2 quickly dropped to normal and remained there. The vision in O. D. improved to normal and the field widened all around.

The second case was of shorter duration: four years in the right eye which was already blind, T + 3, and one year in the left eye, T + 2, and field contracted to 20°. Iridectomy was considered dangerous because of its possible effect on the lens and the probability of choroidal hemorrhage. Trephining was inadvisable because of the congestion. Hence, he had performed double posterior sclerotomy, which was followed by large sacculated subconjunctival effusions that appeared alarming, but were soon absorbed. Tension immediately fell to normal. In three weeks the field in O. S. had widened to 40 degrees and 50 degrees. With proper glasses the vision was normal, 20/15 pt. J–1. He had concluded, from many similar experiences, that division of the sympathetic fibers was responsible for the reduction of tension and the freedom from choroidal hemorrhage after posterior sclerotomy.

Dr. Wm. Campbell Posey said the case is a desperate one. With one eye blind and the other with a field cut almost to fixation, though central vision is normal, the prognosis must be bad. The case does not lend itself to miotic treatment on account of the comparative youth and the social condition of the patient, preventing in all likelihood the persistent use of drops

four times daily for the rest of life. Though iridectomy is my operation of choice in glaucoma, I should prefer trephining here as being less likely to wipe out the small remaining field.

Monocular Diplopia

Dr. William Zentmayer reported a case of monocular diplopia. Miss B., school-teacher, aged sixty-one years, with a history of recurrent conjunctival hemorrhages and retinal arteriosclerosis, had a paralysis of the external rectus of the left eye. Besides the characteristic diplopia of this lesion there was also homonymous monocular diplopia with the right eye closed.

Dr. S. Lewis Ziegler stated that another phase of this subject that Dr. Zentmayer did not refer to, was monocular diplopia from lid pressure on the cornea. During the meeting of the International Ophthalmological Congress, held at Edinburgh in 1894, Dr. Ziegler had heard Dr. George J. Bull, of Paris, discuss this subject with much interest. He claimed that this condition was usually temporary, but was a verity to be considered. He said the French called this *clignement*, which could be freely translated as tarsal constriction.

Bull discovered that he himself was a sufferer from this lesion following his work in ophthalmometry in the clinic of Javal, where he worked for several hours a day with one eye closed. He found that a similar condition existed in the case of artists who half-closed their eyelids while painting and viewed objects through these narrowed slits, thus producing by "nipping" of the lids a rift in the Meibomian secretion or other lubricating substance covering the cornea, as well as in the epithelial surface of the cornea itself. This tarsal constriction was further studied experimentally by passing a hair obliquely in front of the eye. He had the good fortune to observe for thirteen months the eyes of an artist who could make drawings of these monocular doublings, which occurred in each eye separately.

It would seem like a wise precaution, therefore, to at least eliminate this simple corneal lesion before ascribing diplopia to hysteria on the one hand or to organic disease on the other.

A Case of Traumatic Pulsating Exophthalmos Cured by Ligation of the Internal Artery

Dr. Edward A. Shumway reported a case of traumatic pulsating exophthalmos cured by ligation of the internal carotid artery. The patient was a man, aged forty-five years, who had fallen down a flight of steps, four months previously, striking his chin. The following day *x*-ray examination at the Lankenau Hospital showed fracture of the jaw and of the right temporal fossa. A week afterward exophthalmos of the left eye began to develop, and was associated with headache, subjective noises in the head, bruit and pulsation over the eye and the temporal region and paresis of the sixth and branches of the third nerves on the same side. The proptosis gradually increased, producing chemosis of the conjunctiva and moderate tortuosity of the retinal veins. As the symptoms were growing progressively worse, ligation of the internal carotid artery was performed by Dr. John B. Deaver, on March 5, under general anesthesia. On recovering consciousness the patient found the noises in his head had disappeared, and two days later there was a notable recession of the protruding eyeball and freedom from diplopia. There were no cerebral complications, and the man left the hospital on the eighth day after the operation. A later examination showed a difference of only 2 mm. between the position of the eyes, measured by the exophthalmometer; the vision had returned to normal and the external rectus palsy had disappeared. A slight diplopia remained in the upper field, due to paresis of the inferior oblique muscle, but caused no annoyance. There was no recurrence of the bruit or noises in the head, and the patient was apparently cured. Dr. Shumway discussed briefly recent literature on the subject and said the majority of surgeons seemed now to be advocating ligation of the internal carotid as the first operative procedure, with a subsequent ligation of the orbital veins, if the first operation was not successful, before performing any further ligation of the carotid vessels, and he was in accord with this plan.

Dr. William Zentmayer said he would like to take this opportunity to publicly correct a misstatement recently made by a writer that both Dr. Bedell and he had reported instances of cure by gelatin injection. Both of the cases that he reported were treated by ligation of the common carotid, and the cases referred to by the author were cited in the summary of cases in literature.

Since the publication of the paper referred to by Dr. Shumway, he has had another case which was successfully operated upon by ligation of the common carotid by Dr. George Müller. He agreed with Dr. Shumway that operation on the internal carotid is the logical operation. While ligation of the superior ophthalmic vein is a simpler procedure, it is also not without danger from thrombosis.

Dr. Wm. Campbell Posey said he had reported a small series of cases of pulsating exophthalmos operated upon for him by Dr. Edward Martin. Ligation of an orbital vein had been conjoined with ligation of the internal carotid artery. All had had most favorable results save one, in which death occurred several days after operation from cerebral softening. The subject, however, had been an unfavorable one for operation on account of renal complications.

A Case Report of Plastic Operation for Traumatic Deformity of the Eyelids and Face

Dr. Louis Lehrfeld reported that M. C. H., on May 10, 1922, was thrown into a pile of bricks, face first, by a forcible blow on the head, causing lacerations about the right eye, compound comminuted fracture of the nasal bones and possible fracture of the base of the skull. He was treated at Hahnemann Hospital for two and one-half months. Two operations were performed on the eyelids, the first to close wounds and the second to correct ocular deformity.

Patient was first seen by the writer on September 20, 1922, and presented a distortion of right eyelids, in which the palpebral

fissure was inclined down and in and the internal canthus was one-quarter inch below the fellow. The upper lid was fixed and drawn tightly across the cornea, covering the upper and nasal quadrant, and the inner quarter of upper lid border was united to the inner quarter of lower lid, obscuring view of the caruncle and nasal sclera. A dense scar on right wing of nose drew the internal canthus downward. A marked saddle nose, due to the loss of the bridge of the nose, contributed to the lionetic expression not unlike hereditary lues. A large, pendulous, loose mass of tissue under the lower lid obliterated the natural malar-oral crease of the cheek. A K-shaped scar just beyond the external canthus pulled the external angle of palpebral fissure outward and caused a partial ectropion of the lower lid.

On December 9, 1922, a plastic operation was performed at the Garretson Hospital under local anesthesia (1 per cent novocaine). An internal canthotomy was done, cutting down and in to avoid scar tissue present. Another longitudinal incision from the fronto-nasal angle down and out to a point one and one-half inches below the internal canthus, the center of the latter incision meeting the termination of the canthotomy incision. The upper lid was then lifted and its new border sutured high up to meet the plane of the opposite internal canthus. The lower lid was likewise raised to meet the anchorage of the new internal canthus. The conjunctiva was also sutured to the new internal angle of the palpebral fissure and to the skin borders of the canthotomy incision. The result was a widening and horizontal replacement of the palpebral fissure.

On January 3, 1923, a second operation was performed to correct the pendulous mass below the lower lid. A crescent-shaped upright incision was made over the malar region and a large flap of skin excised. The wound was sutured from below upward. As the semilunar incision produced a lesser and greater curvature in the denuded wound, the apposition of tissue permitted an excess of skin at the upper end of the wound. This excess skin was sewed at right angles to the original incision and acted as a stiff support for the ectropion, completely correcting it. Diplopia, originally existent, was completely cured when the upper lid was given

freedom of movement. A specially selected spectacle frame hid the saddle nose, and, in addition to the remodeled eyelids and face, contributed toward a more presentable reconstructed facies.

A Case Report of Foreign Body in the Eyeball

Dr. Louis Lehrfeld reported a case of a young man, D. B., aged twenty years, who was struck in the right eye by a chip of steel while riveting at Hog Island Shipyard in August, 1918. There was a perforating wound of the cornea from the limbus at one o'clock, extending down and out toward eight o'clock, reaching beyond the summit. The eyeball was much injected. *X*-ray examination showed the presence of a foreign body in the vitreous just behind the lens in the lower half of the globe. A magnet extraction was performed by Dr. Burton Chance the day following the injury.

In the absence of Dr. Chance, on military duty, the case was observed by me in the clinic. The eye continued to be irritable, tender, painful, V = light perception. The cornea was smaller than its fellow; the iris was adherent to the corneal scar; and the pupil was occluded and secluded and was attended by a rise in tension.

In November, 1918, two attempts were made to separate the incarcerated iris, both of which operations failed.

On December 1, 1918, an iridotomy was performed with a Ziegler knife needle, incising the iris, exudate and the remains of the lens substance. A V-shaped incision was made, but because of the extent of the anterior synechia, a narrow rectangular opening occurred. Healing was prompt, with rapid subsidence of irritation, tenderness and pain. Two weeks after the operation the patient was refracted by the resident physician, and with a correction similar to that found in aphakic eyes, the vision was recorded as 6/21.

The case is reported four years after the final operation to show that useful vision still remains in an eye, enucleation of which was at one time seriously considered because of the persistence of cyclitis in a shrinking globe.

A Case of Divergence Paresis Following Influenza

Dr. Luther C. Peter presented the case of a young man, aged twenty-two years, who had an attack of influenza five years ago. Since the influenza attack he has suffered from constant headaches. Examination shows a moderately low degree of hypermetropia with some astigmatism. The muscle balance is 7 degrees of esophoria and 1 degree of L. H. at 6 m.; orthophoria at 33 cm. Ductions are: abduction 5, adduction 14. Abduction cannot be improved by prism exercises. The lateral rotations of the right eye are 49 degrees to the temporal side and 41 degrees to the nasal side; of the left eye, 49 degrees to the temporal and 42 degrees to the nasal. Patient's diplopia field shows 9 degrees of separation of the images at 2 meters in the center and to the right and to the left. This is the essential symptom upon which the diagnosis has been made. A tucking of the right external rectus has failed to improve the condition, the diplopia field remaining the same after the operation. The wearing of a 1-degree prism, base out, before each eye, has reduced the esophoria to 5 degrees at 6 m., and converted the orthophoria into 2 degrees of exophoria at 33 cm.

Not a few cases of this condition have been reported in literature. If the diagnosis is correct, it argues for a separate center for divergence, and it would therefore take this function out of the role of a negative quantity, as it is occasionally spoken of as negative convergence. It is an innervational disturbance. From a theoretical standpoint, shortening of one or both external rectus muscles should improve the patient's comfort. In the more severe cases the operation is undoubtedly indicated, as there are no other known means by which the defect can be corrected. In the milder cases, such as the one reported in this communication, it is probable that prisms, bases out, may be prescribed to better advantage. If the occupation of the patient is also prescribed with due consideration for the distance at which he is most comfortable, it is probable that the use of prisms offers the best method of correcting the condition.

Dr. William Zentmayer stated that a differential point between paralysis of divergence and bilateral paralysis of the sixth nerve was that, in the former, images remain precisely the same distance apart in the lateral fields as they do in the middle plane. This is only relatively true, as it follows that in the lateral fields the distance of the object fixed is greater than it is in the median line, consequently the images are wider apart in the lateral field than in the median plane. The statement of Dr. Peter holds good only when the field is taken on an arc and not a tangent. In a case which I have frequently alluded to the patient with this condition is wearing 8-degree prisms, bases out, for each eye, and with these glasses works successfully at his trade of barbering, although his range of single vision with these glasses is not more than 2 or 3 cm.

OCTOBER 11

Practical Aspect of Irregular Astigmatism

Dr. Edward Jackson, of Denver, in opening his paper stated that he found it a source of great satisfaction that he could still count himself a Non-resident Fellow of the College and could not find a more pleasant or profitable place to come when within reach of Philadelphia. He said that he had tried to live up to his opportunities by occupying for several days one of the study-rooms in the College building. With reference to some subjects which seem to be in the outlying territory of refraction, perhaps the best that can be done with them is to show that they are of practical importance, so that they thereby command attention.

He pointed out that in irregular astigmatism the surface causing it curved differently at different points or the index of refraction varied in different parts of the cornea or crystalline lens. A special form is "symmetrical aberration." He gave the case of a young man with negative aberration, so that at the center of the pupil,

each eye was 3 D. myopic, but at the margin of the dilated pupil there was emmetropia or low hyperopia. One eye suffered from traumatic detachment of the retina, and the fundus of the other eye gave an appearance resembling beginning detachment. This was found to be due to differences of clearness of fundus details seen through different parts of the pupil.

In another case, with haziness of the lens nucleus, the eye had poor vision through the undilated pupil, but a sphere 13 D. brought it up to 0.5. When the pupil was fully dilated with euphthalmin and cocain the vision was 0.5 without a lens, the edge of the pupil being slightly hyperopic. In all cases of beginning nuclear cataract some such condition is likely to arise, and cases where the difference is 2 or 3 D. are not rare in elderly people. Cases seeming to show improvement in beginning cataract were quite worthless unless the size of the pupil had been observed and compared for each determination of the acuteness of vision. The presence of symmetric aberration is easily demonstrated in nearly all normal eyes by skiascopy.

Another form of irregular astigmatism is that which gives rise to the "scissors movement" of light and shadow with the shadow test. It is caused by greater curvature of the surfaces toward one side of the pupil than the other. It is common along with regular astigmatism after cataract extraction. A case was cited in which this change of curvature was so great and extended so completely across the pupil that little improvement of vision was possible, although the result of operation was otherwise exceptionally good. The movement generally showed the direction of the meridians of the regular astigmatism present, and the lens that brought the two light areas to come together in the center of the pupil indicated the spheric correction required.

The subjective phenomena of irregular astigmatism depend on the diffusion images that remain after those due to regular astigmatism or spheric errors of refraction have been eliminated by correcting lenses. They include different forms of monocular diplopia or polyopia. They cause the complaints of patients that each letter has "a sort of blur going from it," or "the

letters seem double." The cause of such appearances should be understood by the oculist and explained to the patient. They can be studied and explained by covering part of the pupil and noting what part of the image disappears. For the doubling of a line a card with its side parallel to the line is brought before one edge of the pupil and one of the two images disappears. Any desired quadrant of the pupil may be occluded by a corner of the card, and smaller sectors by a card cut to an acute angle or by the point of a lead-pencil. When parts near the center of the pupil are to be shut off, while the edge is to remain unobstructed, a spot of India ink or a piece of black paper on a glass slide may be used as the obturation. Every case for iridectomy, tattooing or removal of the lens for optical purposes should be studied in this way. The ophthalmologist should be on the alert for this condition. The ophthalmometer may give a clue to it. The ophthalmoscopic examination should include viewing fundus details through various parts of the pupil. For high degrees of irregular astigmatism ophthalmoscopic examination gives most positive, definite and valuable information. Skiascopy should be practised with a small, bright source of light, and from a relatively short distance. Small definite areas of regular refraction should be looked for and measured. Test lenses should be repeatedly tried, using a wide variety of lenses. There is a great opportunity for ingenuity in testing the subjective appearances by shutting off different parts of the pupil, for no two eyes present just the same problems. The practical importance of the subject justifies its careful detailed study.

At the request of the chairman, Dr. de Schweinitz welcomed Dr. Jackson on behalf of the Section of Ophthalmology of the College of Physicians and spoke briefly of his notable achievements in the field of ophthalmology.

Dr. Edward A. Shumway said that, as usual, Dr. Jackson had given something new and worthy of close attention, and it was difficult in the short time allotted to add much in the discussion of the paper. The two points to which he particularly calls attention are symmetric aberration and irregular astigmatism in gen-

eral. As he has well said, aberration is a comparatively common finding when the pupil is widely dilated and may be positive or negative.

Positive aberration is most frequent and may be often demonstrated by retinoscopy, especially in hyperopic eyes, in which, if a small sharp light is employed, a central shadow moving with the movements of the mirror will be present long after the peripheral shadow has been reversed by the lenses placed in front of the eye. This is a point which frequently upsets the beginner's judgment in estimating the refractive errors by this method.

Dr. Jackson's case of negative aberration with a difference of 16 D. is unusual, although myopia is the common accompaniment of advancing nuclear cataract. The practical point is, of course, the necessity for a postmydriatic test in all cases, especially when the refraction of the central visual area differs so markedly from that of the periphery. Patients will not be comfortable if they are not ordered the correction to be used when the pupils have returned to their normal size. The same thing holds true in all types of irregular astigmatism, particularly in conical cornea, to which he has drawn attention.

Dr. Jackson's suggestion of the elimination of the diffusion images which are noticable if the pupil is dilated by the mydriatic, or in a dim light, is a clever one. Passing a card parallel to the second image, especially when two parallel lines are present, will, as he says, cut out one line, which is due usually to the myopic periphery, and will relieve the patient's concern as to the lack of sharpness of the images, even after correction of the regular astigmatism, although many patients are more confused by a scientific explanation than if they are simply told that the eye whose vision cannot be brought to normal is a "lazy eye."

His suggestion that before doing an optical or visual iridectomy, or in the choice for a glass for reading in the presence of opacities of the cornea, a careful search be made not only for clear areas on the cornea but for those which may be best corrected by a lens, should be especially emphasized.

Dr. Edward Jackson (in closing) said that he had nothing to add except that it was one of the very few times that he could not applaud Dr. de Schweinitz.

History of the Study and Practice of the Refraction of the Eye in Philadelphia

Dr. Howard F. Hansell stated that for the past one hundred years Philadelphia has been the home of the professional refractionist. Before the days of specialist the general surgeon made ophthalmology an important part of his work, devoting no little time to the correction of errors of refraction. It is recorded that John McAllister, an optician of Philadelphia, made and fitted a planoconvex cylinder as early as 1828, probably the first attempt to correct astigmatism in the United States, but whether on his own initiative or from a prescription is not stated, probably after many consultations. The men who unconsciously founded the Philadelphia School of Ophthalmology, Dyer, Thomson, Harlan, Norris and McClure, and who established themselves as oculists, placed the study of refraction and the correction of optical defects on a permanent foundation. Under the initiative of these pioneers the correction of errors of refraction was and still is considered of vital importance and was carried on with greater nicety and care in Philadelphia than in most other cities. Dr. S. Weir Mitchell worked in close collaboration with the oculists, and indeed stimulated them to find in the eye causes of nervousness and other reflex symptoms. Evolution from general surgeon to specialist was slow and beset with many difficulties. Finally, ophthalmology was placed on a solid footing and became a recognized specialty. Dr. Hansell concluded the paper with mention of the principal eye dispensaries of the early days and a brief statement of the great value to the profession of the Section on Ophthalmology of the College of Physicians.

Dr. George E. de Schweinitz, after expressing his appreciation of Dr. Hansell's paper made a brief reference to Dr. James Wallace,

for many years associated with Dr. William F. Norris' Clinic in the University Hospital. His intimate knowledge of refraction and of physiologic optics, of which a number of the physicians in the audience were beneficiaries, should not be forgotten in recalling the work and worth of Philadelphia ophthalmologists. Dr. Wallace, he said, possessed a truly "mathematical mind," and few men, if any, among those devoting their attention to ophthalmology were his superiors in this respect. Unhappily, his health declined at a comparatively early period in his career and he was unable to carry on his important work.

Dr. S. Lewis Ziegler said that the early days of refraction in Philadelphia were mostly traditions confirmed by observation and personal contact with some of the active spirits of the time. Credit was given Drs. Dyer, McClure, Thomson, Norris, Harlan, Risley, Randall, Jackson and Oliver as having had much to do with the early history and development of what has been known as the "Philadelphia School of Refraction." As Dr. Hansell has stated, many of the older men gave particular credit to Dr. W. W. McClure an early surgeon of Wills Eye Hospital, as being one of the pioneers chiefly instrumental in bringing about the development of refraction (1) by making some of the earliest tests for astigmatism; (2) by amplifying the fractional units of cylinders below one diopter, and (3) by constructing models of the eye for the purpose of teaching refraction and eye diseases to members of the medical profession who crowded the old clinic room of the Wills Eye Hospital to hear his course of lectures. It was stated that the envy of one of his colleagues caused public teaching to be banned for many years by resolution of the Board of Managers. This early rebuff reacted strangely on McClure and made him very reticent. He never published any of his original work, and hence his fame was soon eclipsed by his more active colleagues. He was the first to refract cases of high hyperopic astigmatism, and his success in correcting the high error of Dr. Thomson turned the latter's attention to this type of ophthalmic work. McClure was later tendered the chair of clinical ophthalmology at Jefferson College, but having acquired a distaste for public teaching he

passed this offer on to Thomson, who was later elected to that position.

McClure's early trial-case was weird but workable, being made up of a variety of lenses both in size and style, in order to fill out the sequence with fractional numbers of one-eighth of a diopter. At a later date he adopted the trial-case manufactured by Ivan Fox. His teaching models of the eye were frequently borrowed by Norris to use in his annual demonstration course at the University of Pennsylvania. It is said that in the early days McClure often laid out his cylinder lenses, fastened them on a sheet of paper, marked the axes and carried them down on a tray to Borsch to be ground. One day, while discussing these problems with Borsch, it was suggested that a protractor would obviate his trouble, and so he added this to his trial-frame, after which it was only necessary to write out the formulas of the trial-lenses in order to have them duplicated.

Dr. Ziegler, said that the trial-cases made by Nachet, of Paris, was the earliest he had personal experience with when he began refraction work in 1886. The axes and lens numbers were scratched on the surface of the glass. This case was supplanted about 1887 by Ivan Fox's trial-case, with the diamond-shaped metal handles that stamped the numbers on them. Dr. Jackson also devised a case of extra small lenses that were manufactured by Queen & Co. Dr. Ziegler's early interest in heterophoria led him to devise a wide metal cell to hold the prisms, as it was impossible to do accurate work with the older types. The first set was made by the American Optical Company, in 1893. The following year he had the Fox Optical Company make up a complete case of trial-lenses mounted in the same type of wide metal cells. These comprised eights of a diopter up to 2 D., quarters to 7 D., halves or 10 D. and single diopters up to 35 D. in sphericals, or fifty-three pairs in all. The same sequence in cylinders reached 30 D., or forty-one pairs in all. The prisms were in pairs to 6 degrees and in single numbers to 40 degrees, or thirty-four in all. A modification of this trial-case was later adopted as the Wills Eye Hospital standard type. He also devised a trial-frame to

hold four of these lenses with wide metal cells, which he has used with great satisfaction during the past thirty years and without a single item of repair. A prism exercise-frame was also constructed with a locking device to hold the metal cells in position, thus preventing rotation of the axis.

Dr. J. Milton Griscom said he only wished to emphasize two general points: Successful refraction is dependent not only on a thorough knowledge of physiology and optics but also upon the exercise of good judgment in determining the relative value of each of the factors which made up a complete examination. It is not enough that one should measure with accuracy the static refraction in every case by means of the retinoscope, trial-case or other method. With this as a basis the other factors, determined by a complete study of the accommodation and the extraocular muscles, together with the age, occupation, general health and temperament of the patient, should each be given careful consideration before a prescription for glasses is given. It is also of the greatest importance in addition that after the glasses are ground and placed before the patient's eyes the ophthalmologist shall personally see that the adjustment is accurate. I believe in most instances where bifocal glasses are uncomfortable it is because they are not properly centered with regard to the upper or lower segment.

When one considers that from 75 to 85 per cent of most ophthalmologists' private practice consists of refraction it would be only reasonable to assume that those specializing in ophthalmology should give a good proportion of their time and effort to this branch of work. In actual practice, however, for several years it has been increasingly difficult to find a student or young practitioner who was willing to devote at least half of his time in clinical work to the study of refraction. A world-wide traveler and trained observer recently wrote that after a survey of world conditions he was forced to conclude that people in general were trying to find a substitute for work. This may explain the tendency evident among certain entirely ethical and able men to advocate refraction by short-cut methods and the unwillingness among students in

general to spend any more time in refraction than they have to. Be that as it may, the fact remains that there is no royal road to thorough and scientific refraction. Patient, painstaking, persistent work is the element without which nothing is possible.

Dr. Edward Jackson said there were two things that have not been brought out here that are of interest to the Section. Dr. Ezra Dyer was working with Donders and Snellen in 1861. On Donders' suggestion Snellen took up the problem of test-types. Dyer, returning to Philadelphia, had printed a card of test-type in 1862, some months before Snellen. Snellen's work was more complete, went into mathematical principles, sifted letters to be used for that purpose, and along with his little brochure on the subject of testing acuteness of vision he put out his test, which became known all over the world. Dyer's type was practically the same as Snellen's type, and he always gave Snellen credit for the principles applied in their construction.

In Philadelphia, at that time, the feeling of certain older men who were in the practice of general surgery, against specialism, prevented Dr. Dyer's work from being properly recognized from the time that he returned to Philadelphia for several years thereafter. He was very much disappointed when he could not get elected to Wills Eye Hospital, a coveted opportunity he only attained years later. The other point is the story of how the first test-case was secured at the Wills Eye Hospital. Dr. Charles Hermon Thomas was the resident at the hospital. One day a woman about forty years of age came in. She had been doing fine needlework for many years and her eyes had given her a great deal of inflammatory trouble. She had gone to one of the most famous general surgeons in Philadelphia, and week after week he had treated her eyes with bluestone without relief. Dr. Thomas, after hearing her story, went to McAllister's and borrowed a trial-case, worked out 5 D. of hyperopia and had glasses made for her. With these lenses she was able to continue her work with comfort for many years. With that story Dr. Thomas went to the Hospital Committee, and the first trial-case was purchased for Wills Eye Hospital. That was the story from Dr. Thomas.

The Technic of Refraction and the Prescribing of Glasses

Dr. S. Lewis Ziegler called attention to the unique advantage to be obtained by a study of the practical methods of different refractionists. He always employs a cycloplegic under forty-five years and often up to fifty, or even beyond when necessary. Homatropin, hyoscin and atropin are his cycloplegics of choice. Homatropin hydrobromate, grain j to ʒss, is instilled night and morning, and every ten minutes for an hour preceding the test, or ten times in all. The hyoscin and atropin are instilled t. i. d. For many years he has used cylinder-fogging in all tests for astigmatism, in order to locate the axis quickly and accurately. The plus cylinder should be at least 1 D. stronger than the supposed astigmatic error. By rotation of the cylinder the vertical lines will lean over to one side or the other and the axis will thus be revealed. This supplants the stenopeic slit. The usual refraction with trial-lenses is then completed. The test-case must be the final arbiter in all cases.

Retinoscopy has proved most useful in children, in illiterates and in proving up the completed refraction by adding sph. + 1 D. and proving the shadows neutral. Javal's keratometer is valuable in differentiating between corneal and lenticular astigmatism or in aphakia. He always utilizes the 6-meter range or a short 5-foot range if the vision is very poor, changing to the long range as the vision is improved by the lenses. His test-card for near vision is based on the Jaeger reading types, to which is added pin-points, needle-holes and a bar of music. Full cylinders are usually ordered, but a slight reduction may be necessary in the high numbers. Cylindrical distortion and anisometropia must be carefully adjusted and the heterophoria corrected. Mixed astigmatism should be corrected by full cylinders crossed at right angles and not by spherocylinders. A slight spherical reduction may be made in prescribing. Irregular astigmatism is a problem by itself, to be carefully worked out and ordered as found. Traumatic astigmatism often falls into this class.

Changes in refraction may be frequent and disturbing. If they cause asthenopia the cycloplegic test should be repeated. In any case refraction should be repeated every year or two in myopia, every two or three years in hyperopia and at similar intervals in presbyopia. All tests should be made two days in succession to confirm the result.

Postcycloplegic tests are made in the majority of cases and the prescription based on all of the units revealed by the various tests. Full spheric corrections are seldom prescribed. Sometimes a reduction for distance is made, but a corresponding increase is ordered for near, thus giving bifocals for young persons. In the high hyperopia of children a partial correction is first ordered and an increase made when the eyes have become accustomed to it.

In hyperopia a reduction of 0.25 D. to 2 D. is made in corrections ranging from sph. + 0.25 D. to sph. + 8 D. In myopia a full correction is ordered up to sph. − 5 D. From that amount up to sph. − 20 D. an allowance of 1 D. to 8 D. is made for near work, or more if necessary. Myopic corrections are prescribed in ground bifocals, with the lower field centered and the upper one deviating slightly. The bottom of the curve is located 3 mm. below the geometrical center of the lens.

For cataract patients biconvex torics are ordered with centers displaced down 3 mm. and the bifocal segments set next to the eye. These are sufficiently prismatic, base down, to bring floor objects up into the field of vision and thus make locomotion more comfortable.

In monocular aphakia the distance spherical is reduced one-half in order to obviate the anisometropia. The regular amount of bifocal is added to both eyes if needed. If one eye is aphakic and the other still cataractous the far and near correction can be ordered in a reversible frame. In young aphakics one eye is sometimes corrected for distance and the other for near. When the average case of aphakia is rapidly neutralized by progressive myopia no glass will be required. Aphakic accommodation is rare but may occur in cases of this character. A correcting lens may then often be omitted.

Heterophoria is measured by the old-fashioned Graefe test, the displacing prism being placed before the right eye and a red glass over the left, with the Greek cross test-object located at a distance of 20 feet. A small black Greek cross, printed on cardboard, is used for the near heterophoric test. The final prism measurement is made over the correcting lenses and may be worn in the prism trial-frame before prescribing. The order for prisms may be divided between the two eyes if over 3 degrees. In correcting hyperphoria the prism is preferably ordered base down because more comfortable. In hyperphoria the total error is prescribed in esophoria two-thirds of the error and in exophoria one-half of the error. The latter correction can often be omitted. Other exceptions may occur. The glasses should be returned for neutralization on the prism scale and for a visé as to fit. At the end of one month the patient should return for examination and adjustment of any psychologic annoyance that a new environment may inflict.

Observation on Refraction

Dr. P. N. K. Schwenk said that in examining the records of 21,812 patients he found 18,268, or 83.7 per cent, came for refractive errors; 440, or 2 per cent, refused drops. These figures illustrate the relative importance of refraction. Good refraction is not common because the students of today shun it as being irksome and tedious. For the best refraction the patient, duly impressed with the importance of getting the most accurate findings, must be comfortable and at ease while the doctor works with perseverence. Complete ciliary paralysis is essential in obtaining accurate results. Since 1887 he had not used homatropin because of its failure to produce complete paralysis of accommodation, its variable strength and the uncertainty of securing the proper drug. Since this time duboisin has been used in all cases under forty-two years of age.

His procedure is to take the family and personal history and note the general physical condition. The visual acuity is recorded

and an external examination of the eye is made. Duboisin (grain $\frac{1}{8}$ to ʒss of distilled water) is then ordered, one drop to be put in each eye the night before and the morning of coming, and the wearing of dark glasses. The drops are to be repeated at noon, in the evening and the next morning, two refractions being done on two successive mornings. To instil the drops the patient is instructed to have the outer one-third of the upper lid pulled up and have one drop of duboisin pass under the lid. In this way the drop will pass over the cornea, thus ensuring maximum absorption. Duboisin, like atropin, causes muscular depression together with delirium, but there is no danger when used as directed with a pointed dropper. The beaded dropper should be condemned, as it gives too large a drop. With cycloplegia complete, the shadow test, fundus examination, refraction and muscle tests are made.

All cases over forty-two years of age are refracted without a cycloplegic, a weak mydriatic being used for the fundus examination when necessary. A correction is given to all patients having a simple hypermetropia of 1 D. or more; below this correction is given full for near work. Any astigmatic error, be it ever so little, is corrected full with as much of the sphere as suits the case. Eserin is not instilled after the cycloplegic, as a rest for the eye is indicated.

The glasses prescribed should have the lenses fixed in the frame to prevent any rotation of the astigmatic axis. In hyperopes the full cylindric with a spheric deduction of from 0.50 D. to 1 D., according to the degree of hypermetropia, is ordered. In myopia a full correction is prescribed in all cases. When the error is 5 D. or more a plus 1 D. bifocal is added, if 8 D. a plus 1.50 D. bifocal, and so on. Low myopes are given a little overcorrection to excite convergence.

Muscles are tested in all cases. Prismatic correction is ordered for an exophoria over 2 degrees or an esophoria over 4 degrees. Lateral exercises are practised in exophoria. Hyperphoria is corrected up to 6 degrees and fusion is obtained. After cataracts have been extracted from each eye there is invariably heterophoria

present which can be relieved by prisms. A sudden increase of hypermetropia in adults should be looked upon as a premonitory symptom of brain tumor. Dr. Schwenk said he had three such cases on record. He has noted an increased in hypermetropia in retinal and choroidal disturbances.

As an instance of rare refraction, Dr. Schwenk gave the following:

O. D. − 16 cyl. axis 10 degrees = 20/50.
O. S. + 6. ⌔ −16 cyl. axis 150 degrees = 20/50.

He wore this correction for twenty-one years, when the following was given:

O. D. − 18 cyl. axis 5 degrees = 20/50.
O. S. + 6. ⌔ −21 cyl. axis 130 degrees = 20/50.

Full correction was ordered with plus 3 D. bifocal.

Dr. G. Oram Ring said that when the chairman expressed his desire to have the speaker outline upon the critical canvas of the hour the subject of present methods for the correction of refractive defects and their associated muscles imbalance, he gave voice to the conviction that we had fallen from our former high estate in the practice of this important division of our special work, which for the average oculist constitutes more than 50 per cent of his consultation hours.

Dr. Ring said that many of the patients who gain entrance to our consultation rooms are of those moderns who feel the driving urge of the insensate craze for speed, that finds its expression in a demand for the very briefest of examinations. The state itself, in this matter at least an unwise guardian, is licensing a group of men called optometrists, not permitted to use cycloplegics, not trained to scientific ophthalmology, but quite sure that cycloplegics are an unnecessary hardship upon the patients and who are using every possible effort to propagate their credo. Of such now practising in our state there are 1100 and upward, over 400 being located in and around the city of Philadelphia. The outcome of such methods you all see far too frequently to here need elaboration.

Dr. Ring said he was in accord with Dr. Schwenk regarding the

desirability of the longer cycloplegic and constantly emphasize its importance, especially to the parents of his young patients. Following the teaching of Dr. Risley, he had for years used hyocyamin sulphate and had never noted any significant constitutional disturbance following its use.

In only one instance in a long hospital service did he recall where a cycloplegic induced a glaucoma. His chief of clinic ordered an assisting intern to write for the patient a solution of boric acid, and she was given a solution of atropin, which she used as an eye-wash. Such a circumstance does not vitiate the thesis. It was simply an inexcusable error.

It is the routine practice in his office, in using a cycloplegic in refracting a patient at or around forty years of age, to invariable take the tension with the tonometer. He did not limit the cycloplegic for refractive purposes to forty-two years of age. In using homatropin he instilled a drop every fifteen minutes for an hour and a half and made the refraction about thirty or forty minutes later. This is often supplemented by the instillation of a drop the previous evening and the early morning.

As to the frequency of examinations in hyperopic cases, there is no hard and fast rule, but he would certainly suggest that the eyes be examined every few years. This is especially important in the lower degrees of hyperopia and hyperopic astigmatism in children during the formative and even more advanced educational period, because of the frequency with which the eye becomes myopic.

A postcycloplegic test is routinely made, and it is of first importance to make a careful muscle study with the new correction in position and base the management of the case from the viewpoint of muscle imbalance upon such findings.

In conclusion he suggested: First, that we emphasize more and more the importance of the cycloplegic if proper refractive results are to be achieved; second, that we increasingly insist upon the longer cycloplegics rather than the more or less unfortunate and often too casual use of homatropin; third, that a properly prepared and carefully applied solution of homatropin is of value, especially

in our adult cases; fourth, that whether or not we have made preliminary muscle studies, that such studies should invariably be made as a part of our postcycloplegic tests, and our judgment as to the muscle management of the case be based upon such examination.

NOVEMBER 15

Dr. George E. de Schweinitz made a brief address on the "History and Activities of the Society afterwards the Section on Ophthalmology of the College of Physicians of Philadelphia." The society was organized November 18, 1890. The Section was established on November 18, 1893, with Dr. William F. Norris, chairman, Dr. Edward Jackson, secretary.

The charter members were Drs. Thomas H. Fenton, H. Ernest Goodman, Howard F. Hansell, George C. Harlan, Francis M. Perkins, B. Alexander Randall, S. D. Risley, John B. Roberts, Edward Jackson, William F. Norris, Charles A. Oliver, G. E. de Schweinitz, Charles Schaffner, William Thomson, Charles H. Thomas.

Monthly meetings, the summer months excepted, have taken place since these dates. During the past thirty-three years, papers relating to practically all the departments of ophthalmology have been presented, a few communications on comparative ophthalmology have been made and considerable attention has been paid to ophthalmoneurology.

Some Extraneous Factors Bearing upon Refraction

Dr. William Campbell Posey said that among these factors should be included changes in the refraction produced by pressure upon the eyeball by swollen lids, enlarged conjunctival follicles, chalazia and lime deposits in the conjunctiva. Pterygium distorts the cornea and often gives rise to high degrees of astigmatism.

Tumors of the orbit may alter refraction, those posterior to the globe making it generally more hypermetropic, those laterally situated more myopic. Increasing hypermetropia has been noted in the early stages of tumors of the optic nerve.

The ciliary muscle is peculiarly susceptible to toxic influences and various degrees of weakness of that muscle may result. Forces ordinarily insufficient to affect the mechanism of the accommodation may, under less favorable conditions, exert a decided deleterious action upon it, for instance, irregularities in behavior of the ciliary muscle from nicotine, when the vitality of heavy smokers is impaired by gastric troubles or impaired functions of the excretory organs. Chronically inflamed nasal sinuses can generate toxins which markedly diminish the range of accommodation, especially if associated with disorder of the general system. Nasal obstructions operate reflexly, as may also dental and tonsillar conditions. The bizarre effect of sugar upon accommodation was dwelt upon; also that of arteriosclerosis. Syphilis has a strong predilection to affect the intraocular muscles, and, in addition to the frank palsies of iris and ciliary muscle which may attend these affections, there are at times less pronounced weaknesses of accommodation which may, however, be sufficient to point the way to the detection of serious involvement of the central nervous system.

Dr. T. B. Holloway stated that of the orbital and lid conditions apt to affect the refraction he believed chalazia were the most frequent. In his experience these lesions were more prone to affect the astigmatism rather than produce such definite changes in the amount of the hyperopia or myopia; while changes, due to conjunctival conditions, undoubtedly have and do cause changes in the refraction, it was most unusual in the experience of the speaker.

As Dr. Posey inferred, the cases of greatest interest were those where the ciliary muscle was affected either partially or completely. This immediately suggests those instances of toxemia following intestinal sepsis, a source which has been much emphasized and one from which the clinical leads are marked, but the scientific

leads are rather short. Much the same may be said about the toxemia arising from adjacent or remote infectious areas.

In at least two papers Veasey has called attention to paresis of the accommodation resulting from focal infections. In two instances Dr. Holloway had seen the same effect following a streptococcus infection; the real interesting part was not so much the clinical manifestations as to how these effects were produced. Is the toxic action on muscles or nerves, or in certain instances are the bloodvessels responsible? In most cases of diphtheria and syphilis the innervation is probably at fault; the same may be said about diabetes; but the muscle is not wholly eliminated. Have these toxins a selective action? Such a theory would be acceptable to some and rejected by others. Two years ago de Schweinitz quoted Flexner as saying we know little or nothing about the toxins of the pyogenic cocci. Does the element of strain or injury, not necessarily severe, enter into the question?

Changes Occurring in the Refraction of Ametropic Eyes

Dr. William Zentmayer gave the following conclusions reached from a study of several hundred eyes in which the interval between the first and the final test was from five to twenty years. Ametropia tends to change in degree even when the error is carefully and repeatedly corrected and the glasses worn constantly. The greatest tendency is toward an increase in the static refraction with an increase in the astigmatism, that is, in compound hyperopia astigmatism the hyperopia tends to decrease and the astigmatism to increase, often proportionately; in compound myopic astigmatism the myopia tends to increase and the astigmatism also to increase. There is no greater tendency for compound myopic astigmatism to change than there is for compound hyperopic astigmatism to change. Simple myopia (rare) almost invariably increases in amount. Simple hyperopia may remain unchanged or undergo an increase or decrease. In mixed astigmatism both of the principal meridians may change in amount and direction. Astigma-

tism is often acquired in hyperopic eyes. Before the forty-fifth year the axis of the acquired astigmatism is as frequently with as against the rule, or, to use the terms Jackson prefers, direct or inverse; but after the forty-fifth year it is against the rule, or inverse, in 85 per cent of the cases. The total astigmatism may be lost, probably in any period, but more commonly in the later decades, and at this time probably represents a transitional stage to astigmatism against the rule (inverse). A change in the direction of the axis of astigmatism occurs in about 50 per cent of the cases.

In 51 of all compound hyperopic astigmatism cases showing a change in the amount of hyperopia or astigmatism there was a decrease of the hyperopia and an increase of astigmatism. In 19 there was an increase in the hyperopia and a decrease in the astigmatism.

In patients over forty-five, in which the average age at which the final test was made was fifty-eight years, in 40 per cent there was a decrease in the hyperopia and in 33 per cent there was an increase in the hyperopia.

In 44 per cent of all compound myopic astigmatism cases showing a change there was an increase in the myopia and an increase in the astigmatism. In 26 there was an increase in the myopia with a decrease in the astigmatism.

Dr. H. Maxwell Langdon said that myopia is retarded by a properly gauged and continually worn correction, but aside from this it seems that correction served rather to lessen discomfort and improve vision than to alter the error. Changes of refraction may be divided into two groups: the first consisting of a small number of congenitally high hyperopic and usually also highly astigmatic eyes which are not prone to very great changes of refraction; and the second, the large mass remaining which may do anything. Astimatism tends to increase, as does myopia, but it is quite impossible to foretell the future in any given case.

The most surprising alterations are those minor ones which occur apparently for no reason in eyes with moderate error—the shifting of an axis 10 or 15 degrees in a year or even less, and only

one eye being affected. Such changes are apt to occur in individuals who are not in robust health and probably represent nutritional changes in the globe, but frequently they occur where the health is excellent. He regarded such changes as a minor cycle of alteration of refraction, whereas those which proceed steadily through the phases which Dr. Zentmayer has described might well be considered as a major cycle of changes.

Dr. B. Alexander Randall said that changes in the refraction of accurately corrected eyes have long been of great interest to us in Philadelphia, where measurements have been made under mydriatic and other precautions which we cannot always recognize in work done elsewhere. Norris and Risley were about the first to certify such changes, although Dor, of Lyons, would "recognize from the cradle as myopic the eyes which the pseudoscientist would set down as emmetropia or even hyperopia," and Macnamarra depreciated full correction of hyperopia as impeding the change toward emmetropia, which he regarded as normal and beneficial. In my study, "Can Hypermetropia be Healthfully Outgrown?" this question, as well as the contrary view as to "hypermetropia acquisita," seemed fairly answered in the negative, and Jackson, as well as myself, has had occasion to note change in the astigmatism of our own eyes measured with reliable efficiency. This latter point especially interested me after I had greatly disgusted Javal by measuring with the ophthalmometer in his laboratory a much higher degree of "astigmatism against the rule" in his cornea than he acknowledged in 1884; and then, four years later seeing him with one eye already lost by glaucoma and the other only partly and temporarily saved by iridectomy. The experimental studies in Pflueger's Clinic, which showed this change in the corneal curves under artificial hypertension, seemed to justify the belief that the like increased curve in the horizontal meridian and human eyes is prodromal of glaucomatous pressure; just as increased curvature of the crystalline lens may mark the swelling of incipient cataract before any striæ of opacity can be recognized. The view presented by Dr. Zentmayer, that such change toward "astigmatism against the rule" is merely senile and not of ill-

omen, is rather tranquillizing, and we must hope that wider and longer observation will confirm it.

In closing, Dr. Zentmayer stated that while it could not be denied that increased intraocular tension might produce an inverse astigmatism, still when we consider that after the forty-fifth year this type of astigmatism is present in 85 per cent of the cases with acquired astigmatism, and also that chronic simple glaucoma is comparatively rare, occurring in only .6 per cent of all eye cases, it is probably a coincidence and not a result of the increased intraocular tension.

The Relation of Exophoria in Early Presbyopia to Refractive Errors

Dr. Luther C. Peter said that routine muscle studies reveal an increasing number of exophorias from the age of thirty-five to that of fifty, most of which can be included in two groups. First, the cases which begin in early adult life and are carried over to the presbyopic period with increased symptoms, and second, "accommodative exophorias" of hypermetropia, due largely to waning accommodation. The paper is concerned with the latter group. A majority of these cases have established for themselves a disturbance in the relation of the meter angle of convergence to the diopter of accommodation. While convergence and accommodation are closely related, and this relation may be a factor in the development of convergent squint, Donders has clearly pointed out that each may work independently in the interests of clear, binocular vision. Cases of hypermetropia, therefore, uncorrected until the presbyopic age begins, are apt to have exophoria for distance with an increased amount of exophoria for the near point.

Steps necessary to correct this type of exophoria are: (1) Careful refraction under a cycloplegic strong enough to uncover all latent hypermetropia. In high degrees of hypermetropia the disturbed relation of muscle balance and accommodation must be corrected gradually by having the patient work up to the full

correction for constant use. A common fault of prescribing opticians, and unfortunately of careless oculists, is to prescribe at this time a full correction for reading and allow the patient to go uncorrected for distance, because they have full 6/6 vision or better. (2) The second step necessary is careful training with prism exercises and the use of the stereoscope. The latter instrument acts as a stabilizer in patients in whom abduction and adduction are both good, but poorly balanced. This group usually have esophoria for distance and exophoria for the near point.

The technic of prism exercises is of much importance if one hopes to obtain results. Each step in these exercises has been carefully stressed in the paper.

The prognosis in this type of case is good if care is observed in the working out of details as outlined.

Dr. William Zentmayer said that Dr. James Wallace taught him that some of the troublesome refraction cases that he would have would be in patients about the age of thirty-five with hyperopia and who had never before worn glasses. In these patients, in whom the accommodation had been in excess of the convergence, the correction of the hyperopia immediately put these two functions in their proper relation. The relaxation of the accommodation results in a corresponding lessened innervation to the interni, with a resulting exophoria. This complication can be met in one of three ways: By giving the weakest glass consistent with the work to be done; by incorporating prisms with bases in, and by adduction exercises. The last is the preferable method and often gives symptomatic relief without apparently changing the amount of exophoria.

In his experience, when an exophoria develops for distance and there is a much higher degree for near (convergence insufficiency with divergence excess), prism exercise frequently fails, while prisms with bases in are of assistance.

The principle of relieving convergence in cases of convergence insufficiency, and relieving accommodation in cases of convergence excess, is applicable to the correction of ametropia at any age.

Dr. H. Maxwell Langdon said that he had seen two cases with

normal vision in each eye and apparently a normal muscle balance, at least their answers when tested with the Maddox rod approximated orthophoria at the reading distance, but at the near point seemed to have a poor converging power, and attempted prism exercises showed binocular simultaneous vision as impossible, the Maddox rod test on close questioning having been seen with alternating eyes. This may be the reason that some of the other cases found it impossible to see two lights simultaneously and is a similar condition.

Dr. B. Alexander Randall said that exophoria had grown into better recognition in the recent decade, and it is now hard to believe that so few understood it in the past. Risley's study, in 1880, of "Weak Eyes in the Public Schools of Philadelphia," just mentioned by Dr. de Schweinitz, was the first, and his "Eyes of Medical Students," in 1884, the second investigation in which opinion was even sought for, and then mainly as "insufficiency" for the near. Those who made inadequate search for latent hypermetropia were then quite as much interested, like Loring, in "insufficiency of divergence." The teaching of Donders as to the range of relative accommodation and convergence is too often forgotten, and the apparent divergence of myopia and even the concommitant strabismus and hyperopia is sometimes assigned to anatomical configuration of the globe and orbit rather than to the essential yoking together of accommodation and convergence. Yet if every uncorrected hypermetrope is not to be strabismic he must exercise this range and generally learn to increase the negative part of his convergence and hence become potentially divergent. Correction of glasses of hyperopia or presbyopia tends, of course, in this same direction; and I believe that Risley and I were the first thus to point to insufficient convergence as characteristic of the hypermetrope rather than of the myope, as was almost universally taught. My own eyes, with low hyperopia but 5 degrees of exophoria for far and 9 degrees for near, as measured in 1879, were painful examples of this truth and doubtless enforced it early and strongly upon my attention, in spite of huge convergent power able to overcome almost any strength of abducent

prisms. The usual name "insufficiency of the interni" was no slight obstacle to a due appreciation that the muscles themselves were probably unduly strong, yet their faulty correlation with the accommodative activity left them still the source of "muscular asthenopia." Certainly, in Vienna, in 1883, I found such error strongly held.

The Refraction of the Aphakic Eye and the Ordering of Spectacles

Dr. Burton K. Chance described the refraction of the eye from which the crystalline lens has been removed, and noted the physical causes for the failure to obtain the highest standard of acuteness of vision even after the most perfect surgical results. He then spoke of the methods he employs in the testing with trial lenses and the making of the spherocylindrical combinations for both distance and near uses. He exhibited a set of trial lenses, made up of spherocylindricals, which are of a great service in the final estimation, in that they are solid like the spectacle glass and can be placed as accurately as the spectacles can be before the eye. He then recited a number of conditions in which the adjustment of cataract glasses might correct the muscular deviations natural to the aphakic eye, and others in which through faulty alignment of the centers of the glasses annoying prismatic effects might be produced, and, finally, of the imperfections of the spectacles themselves and of the difficulties the aphakic experiences in becoming used to the wearing of such powerful lenses. He exhibited a number of forms of lenses to show such imperfections and sets of the so-called "Katral lenses," which are devised to obviate many of the annoyances cited.

Dr. Burton K. Chance exhibited: 1. A girl, of eight or nine years, from whose two eyes he, a year or so ago, had caused the absorption of congenital cataracts in a single operation on each by the deep incisions of both anterior and posterior capsules, as

advocated by Dr. Ziegler. In this case the two eyes were treated at the one seance, and the child was sent home after a few days. At no time was there the slightest reaction. The pupillary spaces became clear at the end of three or four weeks and glasses were immediately ordered. At the last testing the results were most satisfying and the child had advanced rapidly at school.

2. A young girl, of nine years, who presented a broad pupillary membrane covering the anterior pole of the lens, from which thick strands radiate into the iris. In the left eye a complete cataract had been present, but it was removed by a single operation, as in the first case.

True Synchysis Scintillans Contrasted with Snowball Vitreous Opacities

Dr. T. B. Holloway exhibited a patient with complete detachment of the retina, the whole membrane being studded with deposits of cholesterin, and in addition moderate deposits of cholesterin crystals in the vitreous. In the left eye of another patient, reported as Case II in his original report in 1917, there was an excessive development of well-formed snowball opacities. There were several other patients in whom the condition was less pronounced and might have been available for contrast, but several that were followed up had died, moved or would not present themselves. The difference in the two opacities that had been referred to in previous communications could be readily determined.

The case exhibited was the first true instance of synchysis observed in over 16,000 new cases seen during the past six years, and this disregards the new cases in other clinics from which certain suspicious cases could be examined, these invariably proved to be types of snowball opacities. True synchysis scintillans is undoubtedly a rare intraocular condition, especially so where there is an absence of similar deposits in the retina. Not more than two instances of the later type could be recalled from hospital and private experience. The old statistics bearing on this subject must be wholly unreliable.

Greater strides will doubtless be made in the study of conditions pertaining to the vitreous, lens and other ocular structures when further advances are made in biochemistry, and more attention is paid to this important branch by ophthalmologists. At the present time it offers a splendid and increasingly important field to the younger generation, especially since the corneal microscope with slit-lamp illumination has been added as a valuable adjunct to scientific research.

We have all seen cases where the intraocular examination revealed a "small circulation" independent of sclerosis. Have these cases any relation to ciliary complaints or in fact to other ocular discomfort? Finally, could some of our ciliary complaints be due to the presence of circulating vasoconstricting or vasodilating substances?

1. Favus of the Lower Eyelid. 2. Eye Manifestations of Hydroa Vacciniforme. 3. Early Gumma of the Iris with Late Secondary Lesions of the Body

Dr. Edwin B. Miller said that favus of the eyelid is quite rare and is often mistaken for blepharitis marginalis.

There are but two such cases on record. The case presented this evening is the third reported in which there is involvement of the skin of the eyelid without infection of the cilia.

Stelwagon describes hydroa vacciniforme as a rare vesicular disease, usually seen in boys (with only two or three exceptions); it occurs upon uncovered parts, especially the nose, cheeks and ears. The lesions begin as red spots, discrete or in groups, rapidly exhibit vesiculation and later umbilication; the contents become milky, dry crusts, which fall off and leave small vacciniform scars. Fresh outbreaks may take place almost continuously, and the process goes on indefinitely, at least up to young manhood, when the tendency subsides. Its activity is usually limited to the warm season. The first observation was that of Bazin, in 1862.

Examination of this case under discussion revealed photophobia,

lacrimation and considerable flushing of the bulbar and tarsal conjunctiva. The left cornea showed a narrow band of roughening, about 2 mm. wide, extending along the limbus from 2 to 5 o'clock. This did not stain with fluorescin and presented the picture of multiple phlyctenules. From this band, extending in on the cornea, were several fine, gray striæ having the appearance of striped keratitis. von Pirquet showed negative reaction. Treatment: Atropin and hot compresses every three hours, and the diet regulated. The eye cleared up within a week. There was no recurrence.

Early Gumma of the Iris with Late Secondary Lesions of the Body

J. W., aged forty-seven years, colored, came to our clinic suffering from a growth in the right eye, which had developed within a week. Vision: O. D., fingers at 1 foot. O. S., 20/20.

Examination showed congestion of bulbar and tarsal conjunctiva, with a hazy cornea at the limbus; at 7 o'clock there was a marked staphyloma. Back of the staphyloma, at the root of the iris, was noted a growth about the size of a small pea, which pushed the cornea forward. The pupil was irregular, with several posterior synechia, but reacted in the clear areas to light and accommodation. Fundus view was not obtainable. O. S. appeared normal in every respect. Diagnosis: gumma of the iris. The patient was admitted to the house and referred to Dr. Schamberg's Clinic for Wassermann test and treatment. Antiluetic treatment subsequently administered caused rapid absorption of the growth.

Dr. Greenbaum referred first to the patient with active syphilis and a negative complement-fixation test. He said it was very important to remember that with the new Kolmer modification of the Wassermann reaction a positive reaction always means syphilis, but a negative reaction does not by any means exclude that disease. There are conditions in which the reaction may be negative despite the presence of actual syphilis. It may be and often is negative in congenital syphilis; in the early days of chancre,

and very occasionally, as in this instance, during the secondary state; in the late syphilis the percentage of negative reactions is greater in patients insufficiently treated and the reaction becomes negative and may stay so for a number of weeks; in patients sufficiently treated with previous strongly positive reaction it may likwise become negative, but as treatment is continued, becomes positive again for an indefinite period.

The patient with hydroa vacciniforme is very interesting from the viewpoint of both dermatologist and ophthalmologist. It has been shown that almost all cases of hydroa vacciniforme have a hematoporphyrinuria.

Hematoporphyrin is an exudation product of hemoglobin—in reality an iron-free hematin. It is photosensitive and circulating in the blood. It is presumed to be the basis for the development of the skin lesions. It is not hard to conceive of a similar action on the exposed surfaces of the eye. The striking effect of heat is well illustrated in this child; yesterday morning there were no lesions on the face at all. At noon the child went close to a forge and within two hours the numerous lesions noted had developed.

The child with favus is also very interesting because of its extreme rarity. In this case the causative organism is not the human parasite (Achorion schoenleinii) but the mouse or rat parasite (Achorion quinckeanum). The sulphur yellow crust, which all species of achorion produce, may be seen on my own skin, which I had inoculated some three weeks ago for the purpose of studying certain immunological reactions occurring with the ringworm organisms.

DECEMBER 20

The Relation of the Weakness of the Extraocular Muscles to Depth Perception

Dr. William F. Bonner stated that from an exhaustive study of this interesting subject, he had drawn the following deductions:

(*a*) In patients with premature presbyopia: (1) Exophoria was combined with good depth perception. (2) Esophoria was combined with poor depth perception. (3) Orthophoria was combined with good depth perception except when associated with high latent hyperopia.

(*b*) In patients with normal near vision: (1*a*) As a rule, exophoria was combined with good depth perception. (*b*) Two cases had distant vision and depth perception improved by correcting lenses. (2*a*) Low degrees of exophoria were combined with good depth perception. (*b*) High degrees of exophoria were combined with poor depth perception. (3) Right hyperphoria was combined with poor depth perception, but hyperphoria and depth perception were improved by correcting lens and prism.

The exception of the relation of exophoria and depth perception with premature presbyopia was in a patient suffering from pulmonary tuberculosis, in normal near vision the exception was in a case of epilepsy.

Dr. Peter said that Dr. Bonner's paper has a broader clinical application than that to which he has applied it, *i. e.*, to the "air service." He recalled the case of a dentist, who was under his care for high myopia, who found it difficult to estimate the depth of a tooth cavity. Although visual acuity was brought up to nearly normal by glasses, his exophoria interfered in his work to such a degree that he found himself striking the nerve in drilling into the roots in so many instances, and experienced so much difficulty in filling deep root cavities, that he was finally obliged to abandon the practice of his profession.

Dr. Bonner stated that he had found the greatest difficulties in the esophorics and hyperphorics. This is exactly what one would expect. In estimating the sense of depth binocularly, accommodation and convergence are the most important factors along with the use of the parallax. In the presence of a disturbed muscle balance, therefore, the artisan who is obliged to do very fine work may find himself very much inconvenienced in acquiring a proper perspective and a sense of proportion.

The Tonic Effect of the Sympathetic on the Ocular Bloodvessels

Dr. Adler said that a paper read before this Society last year cited experiments which proved the existence of vasoconstrictor fibers to the ocular bloodvessels running to the eye *via* the cervical sympathetic. Since then a series of experiments were undertaken to determine: (1) Whether the influence of these fibers was tonic in effect, keeping the ocular bloodvessels in a semiconstricted state. (2) Whether the influence of these fibers might be protective to the eye and prevent the intraocular pressure from rising to dangerous heights when the general blood-pressure rose. For a complete description of methods employed and the results obtained the original article will have to be consulted. A summary of the results follows:

When the blood-pressure is raised in an animal the intraocular pressure is increased. If the rise in blood-pressure be slow the rise in intraocular pressure is in direct proportion to it. When we compare the rise in intraocular pressure before and after section of the cervical sympathetic with the same rise in blood-pressure, we note that after cutting the nerve the intraocular pressure goes up higher. The sympathetic is effective, therefore, in keeping sudden rises in general blood-pressure and exerting their full effects on intraocular pressure. This gives experimental proof to the assumption of Magitot and others that there is a mechanism in the eye whereby changes in general blood-pressure can be effectually regulated in the eye.

We find no effect on intraocular pressure of cutting the cervical sympathetic on normal blood-pressures. By our instrument, pressure changes of 0.25 mm. Hg. can be registered, so that if there is any effect on the size of the bloodvessels it is too slight to affect intraocular pressure. On theoretical grounds, however, comparing the innervation of the bloodvessels of the eye and ear by the sympathetic, we believe that there is most likely a slight dilation of the ocular bloodvessels on cutting the nerve, only much less than there is in the case of the ear.

CONCLUSIONS. 1. When the blood-pressure is raised the intraocular pressure is increased. This increase, however, is kept in check by a local vasoconstriction of the ocular bloodvessels through the agency of the cervical sympathetic. This mechanism is a protective one to the eye and prevents sudden changes in general blood-pressure from causing harmful changes in intraocular pressure.

2. This protective action is increasingly effective as the blood-pressure ascends. No effect on intraocular pressure is seen when the sympathetic is cut at normal blood-pressures.

3. In the normal animal, as the blood-pressure is raised the intraocular pressure rises in direct proportion to it.

Dr. Scarlett stated that it would be interesting to note, several months later, the ocular tension of animals who had had their cervical sympathetic nerves sectioned, to see if there was any hypotonus present.

In the clinical observation of 11 patients who had received injuries either to the brachial plexus, the cord or the nerve itself, hypotonus of from 1 to 4 degrees was noted in the exophthalmosed eye. The reason for this condition was assumed to be the marked meiosis, pulling the iris free from the angle.

1. DEMONSTRATION OF A CASE OF BIRTH INJURY OF THE CORNEA
2. AN INEXPENSIVE STEREOSCOPE FOR FUSION TRAINING

Dr. Edward A. Shumway demonstrated a case of corneal birth-injury due to the application of forceps. The child, now a boy, aged eight years, was seen four days after delivery. Forceps had been applied because of rigid perineal muscles, the head having remained in a persistent occipitoposterior position. The opacity appeared two days after birth and the cornea was so opaque that no view of pupil or iris could be obtained. As the opacity slowly cleared, two oblique lines were found running downward and outward, corresponding to probable tears in Descemet's membrane. Under the use of dionin the opacity gradually disappeared, leaving a permanent, diffuse haze of the cornea, with especial condensation

in two oblique lines, situated in the anterior stroma, roughly parallel to two delicate lines on the posterior corneal surface, which corresponded to the position of the tears in Descemet's membrane, originally seen. The child now had 3 D. of myopia and 3.5 D. of oblique myopic astigmatism, correction of which gave only 5/150 vision. The fellow-eye had a plain hyperopia of 2 D. Dr. Shumway reviewed the literature and said the case belonged in the third variety of the forms described by Thomson and Buchanan in their papers published in England in 1903, 1905 and 1907, in which there is a permanent opacity which takes a linear form and is due to tears in Descemet's membrane from pressure of the forceps blade. Very few cases have been reported in America, but more probably would be discovered if all patients with high astigmatism in one eye and defective vision were examined with this possibility in mind.

Dr. Shumway referred also to tears in Descemet's membrane which have been reported in other conditions, such as hydrophthalmos, intraocular tumors, high myopia and keratoconus. In keratoconus, however, such ruptures can only be considered as a cause if the keratoconus appears in early youth. The ordinary form which begins at puberty develops without injury to Descemet's membrane.

Dr. Shumway also demonstrated an inexpensive stereoscope for development of fusion training, which he hoped would fill a need, owing to the difficulty of securing a further supply of Kroll's plates, which had proved the most satisfactory, in his experience, for home training of convergent cases in young children. The apparatus was called the "camerascope," and had been brought out for commercial purposes. Special colored pictures were shown, which it was thought would hold the interest of children, one series to secure simultaneous vision and another for fusion. Stereoscopic photgraphs of animal groups and other scenes would be provided, and the sets would be in the hands of Wall and Ochs, in Philadelphia, either for purchase by the patients or for the surgeon to lend if he so desired. Completed sets would be shown later.

CORNEAL MICROSCOPE AND SLITLAMP STUDIES IN THREE INTERESTING CASES: 1. REMNANTS OF PUPILLARY MEMBRANE ON ANTERIOR CAPSULE OF LENS. 2. PIGMENTED CYST OF THE POSTERIOR SURFACE OF THE IRIS. 3. NODULAR IRITIS DUE TO THE BACILLUS OF HANSEN.

Dr. Luther C. Peter said that the first case presented that of remnants of the pupillary membrane on the anterior capsule of the lens, in a girl of nine years of age, who was the victim of hereditary lues. Antiluetic treatment was administered four years ago. The Wassermann now is negative. Hutchinson teeth were well marked. The ophthalmoscope revealed fine, dust-like deposits on the anterior capsule of the lens. Through the dilated pupil the corneal microscope, with slitlamp illumination, showed one remnant of pupillary membrane on the nasal side connecting two eminences on the second tier of the iris stroma. The center of the anterior capsule was found to be covered with numerous star-shaped pigmented deposits, for the most part triangular in shape, but in many instances with four or five points. From these points there extended fine strands of brownish pigment.

According to Vogt these remnants of pupillary membrane obtain their pigment from the blood supply. The anterior vascular tunic receives its blood supply not only from the central artery but also from the ciliary vessels. P. Toulant (*Arch. d'opht.*, Paris, May, 1923) found 41 cases of these deposits in the routine examination of 350 patients, *i. e.*, in 11 per cent of the cases examined. He believes that these pigmented deposits do not represent either embryonal tissue or debris of pupillary membrane, but regards them as cicatrices of iritis and iridocyclitis, and, in many instances, stigmata of syphilis.

That 11 per cent of patients examined routinely should show these deposits seems to be an unusual experience. The author, however, is probably correct in a measure as to their etiology. They probably are the result of an inflammatory process upon the anterior vascular tunic. This theory would more readily account for the pigmentation which is found in the remnants of

the anterior vascular tunic and the absence of remnants in the posterior vascular tunic.

Further studies, with careful search for the evidence of lues in all forms of persistent pupillary membrane, will be of great assistance in the future in arriving at more definite conclusions as to the exact etiology of these stellate cells. With the ophthalmoscope these deposits have the appearance of dots, and their exact character cannot be made out excepting by the use of the corneal microscope and slitlamp.

The second case is one of a cyst of the posterior surface of the iris which, in the writer's experience, is almost unique. It occurred in a child of eight years of age who was seen in consultation to decide whether the posterior polar cataract present should be needled or let alone.

Through the undilated pupil the ophthalmoscope revealed persistent pupillary membrane on the nasal side of the left eye, a posterior polar cataract and a persistent hyaloid artery which could be followed well back into the vitreous. Through a dilated pupil, in addition to these observations, a brown pigmented body was found to project from the nasal side of the posterior surface of the iris forward toward the center of the lens. The slitlamp examination confirmed and magnified the presence of the pupillary membrane, the posterior polar cataract, which was on the posterior surface of the lens, and a persistent hyaloid artery which floated freely in the vitreous and extended well back into the vitreous body. The pigmented cyst, under a magnification of 24 diameters, was found between the ciliary body under the posterior surface of the iris and the lens, which was decidedly flattened and pushed to the temporal side. Projecting from the main body of the cyst, which had considerable depth, were four finger-like extensions which passed back and hugged closely the posterior surface of the lens, extending almost to the posterior polar cataract. The slitlamp penetrated through the body of the cyst without difficulty, and between the lens and the ciliary body the cyst was of considerable depth, but did not push forward the iris tissue. The iris is grayish-blue in color and the cyst is a rich dark brown.

The patient has not at any time had any inflammatory symptoms and complains only of inability to see clearly. This eye is highly myopic and muscular vision is impossible through an undilated pupil because of the posterior polar cataract. The two conditions which suggested themselves to the author were melanotic sarcoma of the ciliary body and a pigmented cyst of the posterior surface of the iris. As there have been no inflammatory symptoms at any time, and because of the presence of other congenital phenomena, *i. e.*, persistent hyaloid artery, the diagnosis of cyst of the posterior surface of the iris has been made. No change has taken place in the size of this cyst during the four months of observation of the patient. No active measures for its removal are contemplated. The case will be kept under observation, and if progressive the removal of the cyst, with immediate examination as to its character, will be undertaken.

The third case is one of nodular iritis due to the bacillus of Hansen (leprosy bacillus), which is unusual, first, because leprosy is exceedingly rare, and second, because, according to some authorities, the nodules of leprosy do not appear upon the iris tissue. For the courtesy of reporting this case the writer is indebted to Dr. Sigmund S. Greenbaum and Dr. Jay F. Schamberg.

The patient is fifty-three years of age, of foreign birth (Jewish), and contracted leprosy of the nodular variety four years ago. The skin surface is literally covered with nodules, and even the mucous surface of the nose and throat has been invaded. The eye complications are recent. There is no invasion of the skin of the lids nor yet of the limbus, the two common seats for eye complications. Although there is no gross evidence of iritis in the right eye, there are two points of posterior synechia visible with the loupe. There is no pericorneal involvement. In the left eye there is slight pericorneal redness. The cornea is rather hazy and scattered over the iris surface are numerous plaques varying in size from a mere pin-point to several millimeters in diagnosis. These plaques are, for the most part, situated between the greater and lesser vascular circles. Tension is good in both eyes.

The slitlamp examination of the right eye shows a few point-like deposits on the anterior corneal surface and a few pigment deposits with isolated mutton-fat clumps visible here and there. The corneal tissue, for the most part, is clear. The iris shows two points of posterior synechia, one above and the other below. The iris for the most part is healthy and there is little evidence of atrophy. Minute white spots, pin-point in size, are visible here and there. The aqueous is clouded and the circulation of the nutrient fluids is plainly visible through the rather clear media. The circulation is rapid. This relative rapidity indicates a receding process. The slower circulation when visible, is evidence, as has been pointed out by Vonder, Heidt and others, of an increasing process and a rather high degree of vicosity of the fluids in the anterior chamber. In the left eye the cornea is densely infiltered. The entire surface is fairly well mottled with small, opaque areas, and the stroma is more or less opaque. The most characteristic change, however, is the diffuse descemetitis which is peculiar in that it covers the entire endothelial surface from the upper to the lower limbus and the endothelium is visible throughout as a gilded mosaic structure. The iris tissue is atrophic throughout; crypts are almost absent. There is almost complete seclusio pupillæ, the pupillary space being also fairly well filled in by dark-brown pigment. Scattered throughout the iris tissue are numerous white plaques varying in size from pin-points, barely visible, to areas one-fourth the size of the pupil. Some of these plaques are a dirty yellowish-white in color and flattened, and others are elevated, giving the appearance of clumps of snow and studded with dots of very white intensity. They are, for the most part, found between the greater and lesser vascular circles, although a few reach to the pupillary space. They differ materially from the tuberculosis nodules found in a tuberculous iris. In fact, they do not resemble these nodules in distribution, in size, in formation nor in general appearance.

The Wassermann in this case was first positive and later negative. The von Pirquet test is negative. The leprosy bacillus has been recovered from skin nodules and is demonstrated in micro-

scopic slides, which Dr. Greenbaum will present in his discussion. They resemble very much the tubercle bacillus, although they can be clearly differentiated.

The outstanding facts in the case are: first, the very slight degree of ciliary inflammation; second, the diffuse descemetitis and the peculiarity of the distribution of the same; and third, the leprosy nodules scattered upon the iris.

The entire condition is improving under the usual local treatment and the administration of chaulmoogra oil.

Dr. Greenbaum said, since ocular complications are common in leprosy there is little to add to the case presentation except that this patient was treated in the genito-urinary clinic of another hospital for nine months as a case of syphilis, and because of a 4-plus Wassermann test. With the new Kolmer C. F. T. such a false positive does not occur. She has shown general improvement under injections (intramuscular) of the ethyl-esters of chaulmoogra oil.

An Ophthalmic Column for Oculists' Consulting Rooms

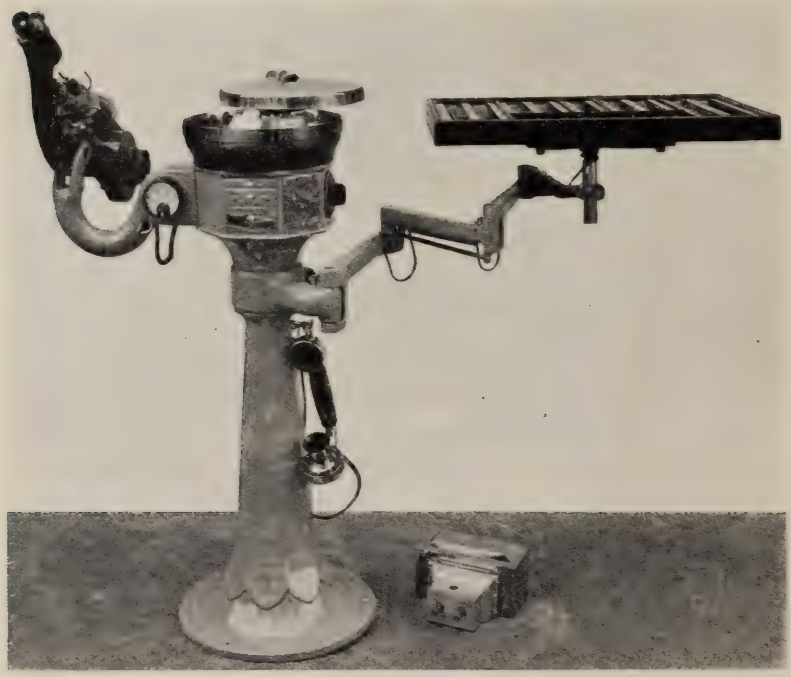
Dr. George S. Crampton exhibited a convenience unit which enables the ophthalmologist to have within his reach most of the equipment necessary for refraction, treatment and minor operations, including means of darkening the consulting room so as to obviate the need of having the patient leave his chair to go to a dark room.

An octagonal cast-iron column supports an extension arm which carries the trial case, which can be moved to any position within a radius of four feet about the column, such as beside the patient for refraction, a meter or so in front for retinoscopy or entirely out of the way during ophthalmoscopy. During retinoscopy the trial case is illuminated.

At the top of the column there is a bronze receptacle carrying eighteen special dropper bottles and solution holders, as well as

receptacles for clean and waste cotton, applicators, wash bottle, etc. A nine-inch disk of heavy white glass in an aluminum frame forms a cover which can be swung to one side as a support for instruments during an operation.

The bronze treatment compartment rests upon an octagonal expansion of the column, which carries the various controlling



An ophthalmic column for the consulting room.

switches and other conveniences, including three small metal drawers for instruments, an electric steam sterilizer, which can be detached when not in use, and a low-voltage rheostat for diagnostic instruments.

There are switches for muscle and room lights and for the visual-acuity cabinet, the illumination of which is under rheostat and voltmeter control. A special switch operates a mechanism in the

acuity cabinet by means of which any desired line or character may be brought into view.

The window shades of the room are automatically controlled by a switch on the column and a motor mechanism at the window, by means of which the shades are raised and lowered to a predetermined height. One movement of the switch darkens the room, while a reversal raises the shades.

Another convenient attachment is the one-hand telephone which may connect with the secretary's desk in the waiting room or with central.

The column was shown with a lensometer attached, but this is not considered as standard equipment.

A Standard Illumination Visual-acuity Test Cabinet having Electrical Means of Exposing the Required Test Characters

Dr. Crampton demonstrated a new visual-acuity cabinet operated by a switch on his ophthalmic column, by means of which any line or character could be brought into view by the movement of a belt of heavy prepared linen three yards in length. The belt was illuminated from the front by means of two long tubular lamps, concealed above and below, and was actuated by a small electric motor, which drew it down from a large shade-roller and wound it upon another roller below.

The illumination of the belt carrying the test characters was controlled from the ophthalmic column by means of a voltmeter and rheostat. The latter was calibrated by inserting the dial of a foot-candle meter through a small window cut in the belt near one end.

An Automatic Device for Controlling the Illumination of Oculists' Consulting Rooms

Dr. Crampton exhibited an electric window-shade controller consisting of a motor-driven drum mechanism which actuated a

pair of toggle switches when the shade was raised or lowered to a predetermined height, thus preparing for movement in the opposite direction when a switch at the operator's working position was turned.

A very small motor operated the drum by means of a worm-gear, thus supplying ample power for pulling the curtain by means of a cord.

This small unit is placed on or beneath the floor near the one or more window shades to be controlled, and after being set for the height of the window or the length of pull, is practically foolproof and obviates the necessity of having a separate dark room and moving the patient from his original position for retinoscopy and ophthalmoscopy.

A New Accommodation Rod

Dr. Crampton showed an accommodation rod made of hexagonal duralumin, 1 cm. in diameter and a little more than 50 cm. in length.

The end of the rod which touches the face or trial lens, as the case may be, was tipped with white celluloid, while the other end carried a ring for hanging.

The rod was calibrated in centimeters with a small stud at the thireenth-inch point.

This equipment was all made by the Lenox Instrument Company, of Philadelphia.

PROCEEDINGS

OF THE

SECTION ON OTOLOGY AND LARYNGOLOGY

DECEMBER 20, 1922

Dr. George B. Wood, the chairman, presented a specimen of an extremely large nasal polyp removed from a mentally defective boy of nineteen years. The symptoms began during the last summer. The soft palate was pushed out to the level of the back teeth and the polyp could be seen below the margin of the soft palate. It was removed by passing a catheter through the nose, for the purpose of introducing a string, in the same manner as for packing the postnasal space. By this a wire loop could be engaged around the polyp and brought through the nose so as to be attached to a nasal snare, the result being that the polyp was easily removed through the mouth.

Presentation of Two Patients with Cancer of the Nose and Throat Treated with Radium

BY DR. RALPH BUTLER

Case I.—J. E., widow, aged fifty-eight years, was admitted to the hospital, November 1, 1921, with a history of profuse watery discharge from the left nostril which followed an attack of influenza in December, 1917. The discharge became mucopurulent and the nose was gradually blocked. No pain, no spon-

taneous bleeding, no loss of weight and no evidence of metastasis. Examination showed a tumor the size of a shelled walnut in the left nostril, protruding into the nasopharynx, and which bled easily. Diagnosis by biopsy, papilliferous adenocarcinoma.

Treatment. 25 mg. of radium in capsule was applied to the nasopharynx and 15 mg. to the left nostril for eight hours. Three weeks later the second application of radium was applied, 35 mg. to the nasopharynx and 15 mg. to the left nostril. Twenty-four days later breathing seemed a little easier and the same dose of radium was used. This was repeated twenty-seven days later and then no treatment for two months.

March 23, 1922. 50 mg. radium applied for twelve hours.

April 11, 1922. 35 mg. of radium applied into the left nasal cavity and repeated one month later for eight hours. This time the mass in the nose seemed to be decreasing in size and the patient felt better.

June 1, 1922. Patient was improved, but complained of a purulent discharge from the right nostril since the last treatment.

June 22, 1922. 50 mg. radium again applied to the nose for eight hours. Condition improved.

Case II.—S. S., male, aged fifty-four years, was admitted to the hospital, November 26, 1919, with the following history: Two years previously patient noticed a deafness in the left ear, which continued to progress. No pain. On examination an ulcerated area was seen in the nasopharynx. Physical examination negative. No metastasis, no loss of weight, blood Wassermann negative.

December 11, 1919. 35 mg. of radium, in capsule, applied for twelve hours. Six weeks later same treatment repeated. No unusual effects except dryness of mouth and throat.

March 18. Examination showed improvement, decrease in size of the growth and gain in weight.

September 15, 1921. Radium was again applied.

March 21, 1922. 15 mg. of radium applied twelve hours.

April 11, 1922. Reduction in the size of the growth, but there was evidence of radium burn.

April 27, 1922. Patient had evidence of radium burn in the nasopharynx and a perforation of the soft palate.

September 14, 1922. Radium treatment applied.

DISCUSSION

Dr. George B. Wood: I have seen one case of squamous-cell epithelioma about the size of a half-dollar, located on the posterior pharyngeal wall, completely disappear after one week's treatment with radium. I saw the patient today, two weeks after the treatment, and only a little whiteness of the mucous membrane, resembling a leukoplakia, could be seen.

Dr. George M. Coates: I saw a case of sarcoma involving the soft palate and lateral pharyngeal wall in 1916. It was incised and radium inserted into the opening, with the result that the growth disappeared, just as in Dr. Wood's case, within a week. No return two months later. After a two weeks' business trip the patient returned with the growth just as it was in the beginning, and it went on to its termination.

Dr. Fielding O. Lewis: I am in favor of the Crosby Green operation when the malignancy involves the ethmoid or maxillary sinus, or both. It makes a very desirable means of applying radium to these regions. This method has been used in the radiological department of the Philadelphia General Hospital. After the surgical procedure, bare tubes are introduced into the involved structure and radium plaques are applied outside. The results have been most gratifying.

Report of a Case of Brain Abscess of Otitic Origin, with Exhibition of Brain

BY DR. GEORGE M. COATES

(*Abstract*)

The patient was a middle-aged man with a history of a running ear on the left side since childhood, but had not had any other symptoms until now. When first seen he had been ill for two weeks. Examination showed the external auditory canal was

swollen closed; no particular signs of mastoiditis. He complained of vertigo and deafness.

Operation revealed a non-cellular type of mastoid, with cholesteatoma in the mastoid antrum and a small cell adjoining. The radical operation was without incident. Was discharged from the hospital in ten days in apparently good condition. Fourteen days following the operation he developed a facial paralysis; no vertigo. Bárány tests showed no response in the vestibular apparatus on the affected side.

Secondary operation five weeks later showed that the labyrinth was necrosed, that the entire wall of the middle ear was necrotic and the anterior wall was removed in one large sequestra. The whole petrous portion of the temporal bone was found destroyed. The patient had complained of severe headaches on the left side before the last operation. Brain abscess was suspected, but the marked destruction of the temporal bone was thought sufficient to account for the headaches. Paralysis of the tongue and pharynx developed on the tenth day following the operation and death occurred a few days later.

On necropsy there was a diffuse, purulent meningitis covering the entire base of the brain. The petrous portion of the temporal bone was destroyed. The pyramid looked normal, but it was found so thin that a knife would puncture it at any point, going immediately into foul-smelling bone. On a cross-section of the brain, three abscess cavities were found in the temporo-sphenoidal lobe, one near the cortex. The whole left side of the brain was softened and flattened out.

DISCUSSION

Dr. George B. Wood: In the army a man was shot in the ear about a year before he came under my observation. He was admitted to the Pennsylvania Hospital with evidence of intracranial pressure. The bullet wound was found to be lying in front of the petrous portion of the temporal bone on a level with the posterior internal wall of the tympanic cavity. The patient developed a series of brain abscesses, and later

developed a hernia of the brain as large as a hen's egg; he died three months later. His whole frontal lobe was gone; there was scarcely any brain substance in it. The parietal lobe was in the same condition, as was also the occipital lobe. Practically one-half of the man's brain was gone and yet he lived for three months. He was able to eat and attend to the normal functions of life until a few days before he died.

Dr. Walter Roberts: The cases that I have seen were similar to the one that Dr. Coates reported, but usually had been tubercular in origin. I had a conversation with Dr. Lewis, wherein was discussed a brain abscess in the frontal lobe secondary to frontal sinus disease, in which there were few symptoms, showing that a great destruction of brain substance can occur without symptoms.

Dr. Fielding O. Lewis: The case mentioned by Dr. Roberts is a young man who had a history of long-continued purulent nasal discharge due to frontal-sinus disease. This was operated on radically. Convalescence uneventful until three weeks following the operation, when he developed signs of pus in the frontal region, swelling, redness and fluctuating of the skin. Later there was a secondary operation. After exposing the whole region of the frontal area a small necrotic area was found in the posterior plate of the left frontal sinus. On curetting this area as much as two ounces of pus was evacuted from the brain. The patient had not had any eye or intracranial symptoms. At this time he is in splendid condition, no rise in temperature, mentally clear and feels good. The cultures from the pus showed streptococci and staphylococci.

Dr. S. MacCuen Smith: One of the most interesting points about intracranial abscesses, otitic in origin, involving the silent area, is the absence of symptoms. In my experience about one-half of the brain-abscess cases are discovered during the radical operation for the cure of chronic suppurative middle-ear disease or mastoid disease. Before the advent of the epidemic of influenza my brain-abscess cases were chiefly the result of chronic suppurative form of middle-ear disease. Since the influenza epidemic the largest number of brain-abscess cases have been of the acute type. When there are symptoms of brain abscess and it is not possible to determine its location, my experience has been that, inasmuch as the vast majority of all brain abscesses of otitic origin occur in the temporo-sphenoidal lobe, one is justified in exploring the temporo-sphenoidal lobe before seeking for the abscess in any other locality. On operating for brain abscess I invariably follow the route of infection. I have never had a case live following an evacuation of pus from any part

of the brain unless this method was followed. I am interested to see in Dr. Coates' specimen that there are several distinct abscess cavities. I do not share the German view that there is always a communication between the various cavities even though this may not be found.

Dr. George Fetterolf: Several years ago, Prof. Behrens, of New York, reported a case of brain abscess, and rather emphasized that one so often has them without symptoms. It was brought out in the discussion, and rather stressed by several men, that a careful study and search as to the disposition of the individual would often show that some changes, in connection with other things, would tend to point out the fact that a brain abscess might be present. There is often a change in the psychology of the patient that might suggest a brain abscess.

Dr. Ralph Butler: I wish to emphasize the point made by Dr. Fetterolf by citing a case which I had some years ago at the Lankenau Hospital. A man had been shot through the upper jaw and the ball was removed through the orbit. Nothing abnormal was suspected in the brain. He became very objectionable in the ward, caused the nurses a great deal of trouble, and his whole disposition was changed. An abscess of the brain was later found.

Dr. S. MacCuen Smith: In regard to the question of aphasia, especially what might be called optic aphasia, a case occurred at the Jefferson Hospital, on whom I operated for brain abscess. The patient had a particular type of aphasia. When he was shown a key, he could not tell that it was a key. He would point to the door and show that it was meant to open the door. When shown a knife, he would show that a knife was meant to cut something. I am of the opinion that optical aphasia is as important a symptom as a changed disposition.

Dr. Coates (closing): My patient has shown both a changed disposition and aphasia. I think that the abscesses shown in the brain were of an acute type, because they were totally without capsule. The final diagnosis was a temporo sphenoidal abscess, but at that time, in view of the extensive destruction of the petrous pyramid and on account of his very poor condition, I did not deem it advisable to search for the abscess. My experience coöperates with that of Dr. Smith, in that they should be opened through the line of infection.

Congenital Atresia of the Nostril

BY DR. WARREN B. DAVIS

(*Abstract*)

Female patient, born December 20, 1920, full term. Family history negative, excepting that the patient is one of twins. Normal in all respects excepting atresia of right nostril. There was a shallow dimple where the right anterior naris should have been, only partial development of the right alar cartilage and more depression of the bridge of the nose than could be considered normal. Most probable cause of the malformation was some continuous pressure on the lateral nasal area during early fetal life by contact with the other twin.

When the patient was five months old, double "Y" incisions were made in the slight depression where the nostril should have been located. The skin flaps thus formed were separated from the underlying tissue. Incisions were made through the soft tissue, keeping the scalpel parallel with the nasal septum as it was carried posteriorly. The atresia was found to extend to approximately a coronal plane at the junction of the anterior and middle thirds of the lower turbinate. The opening was enlarged by the use of a nasal dilator. The skin flaps were placed over the anterior margins of the nostril and a small, perforated, hard-rubber splint placed in the nostril and held in position by a silkworm-gut suture passed through the floor of the naris and tied underneath the upper lip. Previous to operation epiphora had been continuous. There was marked improvement within three days after operation. Contraction of scar tissue after operation was difficult to control in spite of persistent treatment by special dilators. Secondary operation will be required in later childhood. We believe the ventilation secured by the formation of the small nostril will allow proper development of the nasal accessory sinuses and proper function of the nasolacrimal duct.

DISCUSSION

Dr. Benjamin D. Parish: I have seen, about a year ago, an interesting case of congenital atresia of one nostril in a child two weeks old. The right nostril was entirely absent, with the exception that a little tab of skin, which was found patulous, came from the inner canthus of the right eye. On probing this was found to be apparently the lacrimal duct, opening directly into this tube. The question arose whether there was any posterior opening under the posterior superior space and what might be done. The parents were quite willing and anxious to improve the cosmetic appearance, so the lacrimal duct was split and an incision was made along the bridge of the nose and another incision along the cheek, in order to use this flap for the right nostril. The result looked well following the operation, but as it was not his case he was unable to tell what the final result was.

A Radical Operation, Three Years Ago, for Adenocarcinoma of the Ethmoid, Antrum and Middle Turbinate; No Recurrence. Presentation of Patient

BY DR. DAVID HUSIK

(By Invitation)

In reviewing the literature of carcinoma of the inner nose and accessory sinuses he found that the authoritative opinion was that this condition is hopeless. Bloodgood has stated that not one of 18 cases with malignant disease of the nares operated upon at the Johns Hopkins Hospital recovered, and that there is no authentic case on record where recurrence has not taken place before the patient left the hospital. Intranasal carcinoma exists in several forms. The order of frequency is, first, squamous celled; second, cylindrical celled; third, medullary celled; and fourth, adenocarcinoma. Alexander, reporting on adenocarcinoma of the nares, found 1 case aged twenty-two years, 1 aged twenty-eight years and 1 aged thirty-three years. The author's case is thirty-two

years of age. In 200 reported cases of intranasal carcinoma only 21 were undoubted cases of adenocarcinoma.

Jonathan Wright states that he has never seen in the nose and throat, where ablations of various growths are often made at repeated and long-extended intervals, any evidence of a histological change in type from the benign to the malignant in either papillomatous or adenomatous growths. This view is not held by others, who believe that adenomas and papillomas frequently become carcinomatous, and they account for the rarity of adenocarcinoma as being due to the rarity of intranasal adenomas.

The author reports the following case for the reason that the patient is free from recurrence three years after operation for removal of an adenocarcinoma involving the middle turbinate, antrum and ethmoid on the left side:

A. W., aged thirty-two years, reported to the University of Pennsylvania Hospital, in May, 1919, complaining of left-sided nasal obstruction and discharge. Examination showed the left nostril filled with a large, grayish, irregular, polypoid mass covered with a mucopurulent discharge, boggy to touch and evidently attached to the middle turbinate. Transillumination showed a dark area below the left eye and over two-thirds of the internal aspect of the antrum. Under cocain adrenalin solution enough of the growth was removed to establish nasal breathing.

The patient disappeared, but returned to the hospital November 2, 1919, with a severe left-sided nasal hemorrhage. Examination at this time showed the left cheek and eye more prominent, and that he had developed left-sided deafness.

The following biopsy study by Dr. Allan J. Smith was given: "The general appearance of the histological structure would tend to its classification as an infiltrative adenocarcinoma with origin in a mucus-secreting gland, such as occurs in the mucous membrane of the nose." *X*-ray report showed disease of the antrum, anterior and posterior ethmoids. 50 mg. radium in capsule was directly applied to the growth every seven days and *x*-ray treatment was given by Dr. Pancoast with little effect. On December 18, 1919,

radical operation for removal of the upper jaw was performed and the ethmoids were curetted.

Several days following the operation an acute otitis media developed, which promptly cleared up. In March, 1920, there was seen evidence of recurrence in the region of the superior turbinate, which disappeared after applying 50 mg. of radium. The biopsy report was adenocarcinoma.

DISCUSSION

Dr. Fielding O. Lewis: Dr. Husik should be congratulated upon the splendid result, and I think that the use of radium and *x*-ray was very timely.

Dr. George H. Coates: Dr. Husik, I think, quoted Dr. Bloodgood to the effect that these cases never recover. The Boston doctors have shown that they do recover. Their plan is the open-face method, keeping the antrum open until the possibility and probability of recurrence are over.

Laryngeal Gumma in a Ten-year-old Child

BY DR. JOSEPH D. SEIBERLING

(By Invitation)

(*Abstract*)

Evasive symptoms, the etiology of which is obscure, should direct one's attention to the probable factor as being syphilis. This is true especially just preceding or during adolescence. I could do no better than to refer to Hunter's *Commentary on Syphilis*, in which he states, concerning laws of evolution of hereditary syphilis: "It is certain that an infant may pass through the secondary stage of inherited syphilis without ever presenting any symptoms. Those who have shown no symptoms in infancy may yet suffer in later life. It is clearly to be understood, however,

that where this happens to adolescents they suffer from a class of symptoms wholly different from those of infancy. It is not that the secondary stage has been delayed, but simply that it has passed through without ostensible disturbance. After a period of latency, there will in many cases come a group of very peculiar affections involving principally the sense organs."

D. S., aged ten years, Russian parentage, born in U. S. A. Good health since birth. One uncle died of tuberculosis and another of cancer. Father, forty-eight, living and well. Served in the Russian army. Two years ago had swelling of the thumb, which disappeared under mixed treatment. No Wassermann blood-test at that time. Denies any knowledge of visible lesion. Has had occasional headaches, also fullness in the head. Blood Wasserman plus two. Mother, forty-seven, living. Had thirteen pregnancies; the fifth, sixth and eleventh terminating at ten weeks, three and one-half and six months respectively. First child died of hemorrhage. Third child died three weeks after birth from infection following circumcision. Eighth child died of scarlet fever at three years of age. Ninth child died of diphtheria at one year. Six children living and well.

The subject of this report is the twelfth child. History of present illness: Six months ago, after returning home from school, noticed hoarseness; no sore throat. The hoarseness progressed until the voice became only a whisper. Was advised by family physician to have tonsillectomy. Reported to the Polyclinic Hospital in September, 1922. General physical examination negative. *X*-ray examination showed no metastasis in the lungs. Blood count shows: Erythrocytes, 4,610,000; leukocytes, 9300; color index, 90. Blood Wassermann positive plus four. Examination of the larynx showed left ventricular band and cord infiltrated with neoplasm, which projected beyond the median line, somewhat irregular but smooth in contour, slightly seminodular and apparently fixing the cricoarytenoid joint. Right vocal cord slightly congested but otherwise normal and moves freely. Glottic chink narrowed to about one-third normal. Slight restriction of

suprasternal notch on inspiration. September 21, received 0.45 arsphenamine intravenously, and this treatment has been continued once weekly up to the present time. Retrogression of the mass has been rapid.

DISCUSSION

Dr. George B. Wood: This is an extremely interesting case, gumma in children being very rare, and especially gumma of the larynx.

Dr. Walter Roberts: In my experience the blood Wassermann reaction is often useless as a diagnostic measure. I have used potassium iodide as a therapeutic test in the diagnosis, and am of the opinion that good results are often obtained when little anticipated by the use of ascending doses of potassium iodide, although repeated Wassermanns have been negative.

Dr. Curtis C. Eves: Dr. Roberts' remarks call to mind a case treated some time ago, a man, seventy years old, with neoplasm of the larynx. Three negative blood Wassermanns. No specimen had been removed for diagnosis. Patient was treated with radium and *x*-ray with little improvement. Later the patient's family physician administered potassium iodide for some asthmatic symptoms and the growth began at once to disappear.

Dr. Sieberling (closing): The patient's Wassermann is still plus four.

A Case of Cerebrospinal Rhinorrhea

BY DR. JOHN EDWARD LOFTUS

(By Invitation)

(*Abstract*)

The author, after giving a complete review of all published cases and literature of cerebrospinal rhinorrhea, the comprehensive and thorough data as to the etiology, pathology, symptoms and diagnosis, reports the following case:

A woman, aged forty years, was first seen January 5, 1922, after being treated three months for sinusitis. She was complaining of a constant dripping of clear fluid from the left nostril for the past three months. The patient has always been very stout. Present weight, 240 pounds. No history of any head injury. Present condition began October 23, 1921, after a very hearty laughing attack, which was followed by severe sneezing. Patient was aware of a clear, limpid fluid from the left nostril, associated with a frontal headache extending to the vertex. The headache has been constantly present, but diminishes somewhat when the flow increases. One-half ounce of fluid escaped in one hour. The fluid was clear, odorless, tasteless and without sediment, with an alkaline reaction. The flow of the fluid is greatly altered or increased by the position of the head. When the head is dropped forward the flow increases; when the head is in the upright position the flow is less; when lying in bed with the head hanging over the edge, face downward, there is a drop of fluid every two seconds. During sleeping hours the flow is the same as during the day. The constant swallowing of the fluid at times has caused severe diarrhea. On three occasions the patient states that the fluid escaped from her left ear; that it came out in gushes and lasted a short time. This was not seen by the author. The fluid is also increased when the patient works, climbs steps, does any severe exercise, or when she becomes angered and excited.

Examination of her nose revealed a sharply deviated septum to the right. There is a spur at the base of the septum on the same side, which touches the inferior turbinate. Tonsils are of average size and apparently normal. The sinuses were negative. The author was unable to determine definitely where the fluid made its exit, but was of the opinion that it came from the posterior portion of the roof of the nose and not from the middle meatus. Examination of the ear was negative. Wassermann reaction negative. On examination, the escaped fluid showed the same composition as that of a normal cerebrospinal fluid. Eye examination had no bearing on the symptoms. Complete neurological examination showed no evidence of organic disease of the brain.

Conclusions. Patient became so depressed and melancholic that it was deemed advisable to send her to the hospital for a rest cure. After being in the hospital two and a half months the flow of fluid from her left nostril entirely subsided. Two weeks following the cessation of the flow she was suddenly taken with a chill and a rise of temperature of 104°, associated with this was numbness of the entire body and severe headache. Two days later she went into a semicomatose state with alternate periods of lucidity. The following day she developed a facial paralysis and tremors of the entire body. Two days following she developed auditory-nerve deafness and three weeks later died of acute encephalitis.

DISCUSSION

Dr. George Fetterolf: To my mind this was one of the most notable presentations I ever listened to; for carefulness of study and for accuracy and clearness of presentation it is particularly notable.

Dr. Warren B. Davis: I was asked to see the patient, while in St. Agnes' Hospital, because of her pain, which had developed in the left ear. That examination of the ear was negative. There was one point which Dr. Loftus had not mentioned, and that was the possibility of a fracture having been made at the time of a submucous resection, which had been performed some time before, but I was unable to determine whether this was the case. The upper part of the resection seemed to have been done fairly high. There was a firmness of the septum where Dr. Loftus said there was still a ridge. There was also a slight palsy on the left side at the time of his examination.

Dr. George B. Wood: Had a postmortem been secured?

Dr. Loftus (closing): Unfortunately, due to the pathologist, a postmortem was not done. I had been given the privilege, but the proper person was not around at the desired time. Regarding the escape of cerebrospinal fluid from the ear, in reviewing the literature I found that Escot reported a case of cerebrospinal fluid from the left ear following a submucous resection. No other cases were found in the literature.

A Series of Colored Paintings Illustrating, in Progression, the Pathological Condition of the Membrana Tympani

BY DR. JAMES H. MENDEL

(By Invitation)

(*Abstract*)

The author showed in succession painted illustrations, two hundred times larger than the normal size of the drumhead, taking up in succession the normal ear-drum and the various pathological conditions which are found. The plates represented an enormous amount of work and careful study made by the author. The minute details that were brought out and explained were both interesting and instructive to all members present.

JANUARY 17

The Nature of the Influence of Focal Infection and the Measures Necessary to Meet it

BY RALPH PEMBERTON, M.D.

As an internist, I feel a compliment that any of you should be interested in the work that my associates and I are doing at the Presbyterian Hospital. Dr. Fetterolf has asked me to talk on the question of focal infection, but it seems to me that it is necessary to realize that in studying focal infection one is of necessity studying, or one must in part study, the condition which it produces. There is small virtue, I take it, in studying merely round-cell infiltration around a localized abscess, and what we are most interested in, at the Presbyterian Hospital at least, is what that process does elsewhere in the body.

We have studied focal infection incidentally, in a sense. For some years we have been interested in the topic of arthritis, which, as you know, is often brought about by focal infection, perhaps more often than by anything else.

What I will try to show you tonight is something of the pathology back of this peculiar syndrome which focal infection may bring about. I did not fully know until we began our studies that arthritis is only one of a great number of conditions which arise from the same premises. There are a great many neurological conditions associated with the syndrome of arthritis, many skin diseases, certain eye conditions, etc., so that it is a very large field, and I think there are very few topics in medicine of wider extent or of more importance than this vague field which so interests us.

Our largest experience in this regard was in the Army, where we had the opportunity of studying about 400 cases of arthritis, the largest number that has been studied under controlled circumstances. While we are concerned primarily with arthritis here, this is merely one manifestation of the syndrome which foci bring about, and many varying conditions also come under observation. The principal exciting cause in this large number of selected young men, I may say in about 60 per cent of them, was exposure to cold or wet, or both, and incidentally in their histories tonsillitis precipitated only $3\frac{1}{4}$ per cent of cases. The second exciting factor was dysentery. It will be interesting, I think, for you to note, as it was to us, that of these 400 cases, 184 of them, 46 per cent, recovered in the presence of a demonstrable surgical focus. That is to say, a larger number of men came to the hospital who were either well or getting well, or got well as the result of the various accessory forms of treatment, than were cured as the result of the removal of the foci which they presented. I do not want you to think that I am minimizing the importance of focal infection. I am keenly conscious of the importance of it, but I think it is important also to see what the limitations are in this field. I think this series of cases observed in the Army is of very real value, as the disease was caused by conditions of war and represented,

in a sense, an experimental production of it. More got well through the various accessory activities of the hospital than got well through attempts to remove the foci which they presented. That is not typical of civil life, as we all know. These cases were in young men with fine powers of resistance. They were selected subjects and the situation was unusual, but the principle under discussion is, nevertheless, well illustrated. In contrast I can say that only 8 per cent got well, as far as we could analyze it, as the direct result of the removal of focal infection.

I want to call your attention briefly to some of the pathology which we find in studying arthritis. I would like to say again that I do not want you to think that we take an attitude which is negligent of the importance of bacteriology. We feel in our clinic that the bacteriology of arthritis has been very thoroughly and successfully studied by a large group of men. As far as arthritis is concerned, however, and even more in nervous diseases and some other branches of this broad syndrome, there is small evidence to indicate that bacteria at other parts of the body are important or large factors in producing the disease at the given site. For example, if a man has a bad tonsil and a bad knee, it does not mean that he necessarily has bacteria in the knee merely because he has them in his tonsil. I think one should have a perfectly open mind regarding this topic, and we try to keep away from bias.

We have endeavored to study some of the pathology of this syndrome from the standpoint of biochemistry, and began, for a number of reasons, with a study of the so-called basal metabolism, namely, the amount of heat liberated by the body per unit of surface area per hour. Of 29 cases, 20 per cent had a basal metabolism a little bit below normal. Incidentally, it may interest you to know that there is no disturbance of the blood nitrogen and the renal function in the usual sense. One hears a great deal in this syndrome of cutting out meats and that sort of thing, but there is no evidence to justify it, so far as the meats, *per se*, are concerned.

Another topic that we studied at considerable length was the

question of the so-called sugar tolerance. This is a very much abused test and is not specific for any one thing. It merely shows that something is going on back of the surface pathology. (Illustrates with charts.) If you give a man some sugar or glucose by the mouth and examine his blood for sugar before you give it, and then again about half an hour or an hour afterward, you will find that the sugar increases in his blood. The degree of increase is of interest. If it increases a great deal we say that he has a lowered sugar tolerance. If you examine a large series of arthritic cases, as we did, you will find that for some reason these people cannot remove as promptly as normal the sugar which gets into their blood from the intestinal tract after they are fed. (Chart illustrations.) The fact is that in chronic cases of arthritis and certain neurological conditions, various skin diseases, rheumatism, neuritis, etc., this disturbance in the blood chemistry may persist for a long time after the removal of what we regard as the causative factor.

Some of you are going to ask me when you should take out tonsils. I cannot tell you, but I can say that here is perfectly graphic evidence that there is a definite dislocation of physiology which remains for some time after the apparent cause is removed. This is part of the reason why many cases do not at once get well.

On many grounds which I will not take your time to relate here we have thought that this underlying disturbance might concern some phase of oxidative metabolism. We have studied, therefore, the respiratory function of the blood. Dr. B. M. Hendricks, who was with Dr. Alonzo Taylor at the University, and Miss Crouter and I have become much interested in this field and have come across some suggestive data. We find that there is a little difference in the amount of oxygen in the peripheral venous blood of the arthritic as compared with the normal individual—that is, the arthritic may have a higher "percentage saturation of oxygen." That means that there is a less amount of oxygen in the veins, under the condition in which we take it, as the blood comes back from the periphery, than there is in the normal individual under the same circumstances. These figures run in

the neighborhood of 46 or 48 for normals under these conditions. In the arthritic they run a little higher, and some cases show a very much higher figure, 80 or even 90 per cent. Furthermore, when you give such a man the glucose tolerance test you find not only that the sugar is too high, but that during the test he has more oxygen in the blood than he ought to have. If the oxygen is in the blood it is evidence that it is not in the tissues, and it suggests that this man is not getting the complete oxidation within his tissues that he ought to have. We tried to explain this on the basis of chemistry, feeling that there might be some chemical combination by which the oxygen was unduly bound, but we could not do it. So we were driven to the thought that perhaps there might be some change in the blood flow which might be so fast or so slow that the sugar could not be removed. We have spent a great deal of time trying to work this out by means of "hand calorimeters," and have observed that the arthritic tends to have a lower blood flow in his periphery, or in his hand, than the normal individual has. The corollary to this is, as you all know, that the arthritic subject is at his worst in the early morning but improves very much as the day passes and he becomes more active. It is easy to see that precisely as he improves during the day his blood flow goes up. Some patients have said to me that their cup of coffee put them straight. That was the first thing that caused them to mend for the day. The caffein in the coffee presumably caused a lengthened circulation.

Another agency which is of value in these conditions is that illustrated by hydrotherapy, external heat, etc. The subject of sweating and the nature of the influence of hydrotherapy upon the human body is almost untouched, though Bazett, of the University of Pennsylvania, Haggard, of Yale, and Talbert, of Chicago, have done some work in this connection recently. Dr. Cajori, Miss Crouter and I have become much interested in this field and have carried out a great many experiments.

As far as we can see from the literature, it has not before been noticed that if you bake an individual the sweat induced will change its reaction, or, technically, hydrogen-ion concentration,

as the bake progresses, whatever that reaction may have been at the start. It is interesting to recall incidentally that the amphibia, such as the frog, normally respire through their skin to a large degree. When sweating begins there may be either an alkaline, neutral or acid flow. In the majority of cases it falls roughly around the neutral point. As the bake progresses this sweat becomes alkaline, or if it starts alkaline it becomes more alkaline; if it starts acid it becomes less acid or neutral and may even become alkaline. If you study the arthritic, much the same thing happens, except that there are some marked exceptions in which the sweat is exceedingly acid and remains so throughout. So there is large evidence to indicate that the syndrome in which we are interested, of which the arthritic is perhaps the most easily studied exponent, departs from the normal in this respect.

Now, *apropos* of the skin function, to which I called your attention a minute ago, I want to show you something else that takes place when this bake is carried on. If you take a man's arm, surround it with a rubber bag and then bake the subject, you will find that there is given off from the skin carbon dioxide. This is a known fact, although not very widely known, but interesting in that precisely as the arthritic or any individual tends to lose the acid nature of his sweat at the outset, so at the same time he loses carbon dioxide through the sweat of his arm, and it increases in tension in the bag as the bake goes on. We are working on this problem now, and I mention it to give you some idea of what is taking place; but there is some very strong suggestion that part of the benefit which is derived from this measure has to do with the elimination of carbon dioxide through the sweat. Furthermore, it is eliminated also through the urine in increased amounts.

Carbon dioxide is the end-product of combustion in the body. There are probably several acids which contribute to the reaction of the sweat, but it is almost certain that carbon dioxide is one of them.

On the basis of the suggestion as to the metabolic difficulty which these individuals suffer under, I want to speak to you of treatment.

There are many measures which improve this general syndrome. When a person has an obvious focal infection bringing about the disturbances under discussion, it should, if possible, be removed, but the situation does not always clear up when that takes place. Then what can we do? Fortunately, Nature has her way of meeting this difficulty. If this were not so, the boys in the service would not have gotten well in the manner described.

Furthermore, there would be no justification for the many sanatoria and spaas which depend upon hydrotherapy, mineral springs, climate and the like, and have existed in one form or another since the days of the Romans.

I have spoken several times of metabolism, or the chemical interchange which goes on in our bodies all the time. It may do so at the normal rate; it may do so at a rate slower than normal, as in myxedema; or it may do so at an increased rate, as in fever, exophthalmic goiter, etc. In general, the agents which hasten metabolism are of value in arthritis and other manifestations of the syndrome under discussion. One of these agents is hydrotherapy.

There is no question that external heat, and exercise, and massage, and measures of that sort have a very profound effect upon the body metabolism. This effect may be local, as in an arm, or it may be systemic. Arsenic has the same effect. Radium is another substance which is becoming more and more used for the same reasons. The iodides probably have some effect in the same direction, and thyroid extract certainly has.

Again, if an individual suffering from arthritis, for example, be subjected to tonsillectomy because of a bad pair of tonsils, there is not infrequently noticed on the second or third day after the operation great improvement or entire relief in his underlying trouble. I venture the guess that many of you gentlemen have noted this relationship. The end-result to this individual may or may not be good, but the temporary improvement arises from independent causes. They are chiefly two, namely, a great stimulus to his metabolism occasioned by the operation as a whole and the nearly complete curtailment of food for twenty-four

to forty-eight hours, which involves a reduced load upon his body chemistry. I will refer to this later.

By the same token, the use of non-specific protein injections in arthritis, chronic sinus conditions, etc., depends upon the increased metabolism which is induced by the rise in temperature. Du Bois and others have shown that during such a chill, which is closely comparable to the malarial chill, there is a large combustion of liver glycogen.

There are some other conditions whose influence on metabolism is perfectly clear-cut. If a woman has an arthritis and undergoes pregnancy, in a great many cases she undergoes also practically an entire amelioration of her arthritic syndrome. During that time, of course, it is well known that the metabolism may increase as much as 30 per cent. I would like, therefore, very much to have you get something of the concept we are now entertaining that this great syndrome under discussion may be regarded as a sort of equation. On the one side are the factors making for a difficulty in the oxidative process. On the other side are the measures which influence metabolism. It is possible to meet the difficulty going on one side by appropriate influences on the other side, such as treatment by massage, hydrotherapy, exercise, certain drugs, etc. There is another way, however, in which these individuals may be benefited.

There are two kinds of metabolism, namely: Endogenous, or that which originates in the body and constitutes the chemistry of the tissues uninfluenced by external factors; and exogenous, or the metabolism of the food which is taken in from without. We have hitherto discussed measures influencing the former. It is obvious, however, that by definition it is possible importantly to influence the latter by alterations in the food intake. If our hypothesis, however, is correct that this general syndrome under discussion is one in which the intensity of metabolism is lowered or in some way obstructed, it should follow that measures directed at reducing the load upon the metabolic processes should have value, and, indeed, this proves to be the case. It is possible by lightening the metabolic load in some arthritics so to improve the

metabolic processes that very graphic results follow. You know if you eat more food than you can take care of you will have difficulty in utilizing it. Some persons can take about 3000 calories, and if they lead rather active lives they can get away with it. We had a man in the Army who had a very bad case of arthritis and ate a total of 3800 calories a day. He had had some foci removed in the way of dental infection, but he ate a lot of chocolate and fudge and over twice as much food as a man needs to maintain his nutritional equilibrium when remaining in bed. We put him on 2000 calories, and this is what happened: In three days after we diminished his diet he was out of bed; in twelve days he discarded his crutches and walked about without pain. He left the hospital after five months, entirely well. Fletcher, of the University of Toronto, has just corroborated our work by further experiments: He has taken at random 100 cases of arthritis with bad teeth and other foci. They were put on a diet of a few hundred calories at the start, later increased, and he found that of this group 8 per cent made a complete recovery with no other treatment but cutting down their food; 43 per cent were distinctly improved. He has taken another series of 50 and studied them in the same way with exactly the same results.

Another interesting phase of physiology which throws some light upon the mechanism of arthritis and associated conditions, is that opened up by study of the effects of high altitudes. For example, during the recent expedition of Barcroft to the Andes, living at 16,000 feet, it was noted that under these conditions of anoxemia the development of sore muscles, known popularly as Charley Horse, developed much sooner after exercise than it does at low levels. They also observed as the result of adaptation to these altitudes an enlargement of the finger-ends comparable to that seen in hypertrophic pulmonary osteoarthropathy, and it is well-known that in this condition sometimes the actual bones themselves become involved.

One can also see some of the symptoms of this syndrome exemplified by aviators, particularly headache, fatigue and certain mental disturbances. It may interest you to know, incidentally,

apropos of these difficulties in oxygenation, that the joints are structures having little or no blood supply of their own.

The work of Krogh and others has opened up recently a very interesting field in relation to the capillary-bed intermediary between the arterial and the venous systems. It is clear from his work and that of other physiologists that we have been neglecting a very important part of the vascular system. There are many reasons to believe that some of the benefit from hydrotherapy and massage in the conditions mentioned above is due to the influence of these measures upon the smaller bloodvessels.

Without unduly taking your time, it may be said that there is evidence to indicate that such products of proteolysis as may result from focal infections conceivably influence this fine vascular bed, and, indeed, the finer bloodvessels in many parts of the body. This conception of one of the causes of arthritis fits in well with the viewpoint, reached on other grounds, that the disease is referable in part at least to changes in the supply or functions of the blood.

There is a group of workers in this country who have been long and profitably interested in the study of chronic invalids of various kinds, including the arthritic, and that is the Boston school of orthopedists. I heard someone say tonight before I came here that the person who most frequently applied to him as having bad tonsils was the patient who was in bad general health, although lacking any specific complaint.

The orthopedists have taken a long-range perspective of such cases as machines in which many mechanical features may go wrong and be caught up in a vicious cycle. Many departures from normal physiology arise from an invalidism secondary to the original factor, be it focal infection, exposure or what not. The aim of these workers, therefore, has been to improve this faulty physiology along broad lines by the adoption of postures and exercise which aim to "hyper"-correct. Some of their measures are directed at improving the thoracic capacity, widening the costal angle, increasing the range of the diaphragm and overcoming the enteroptosis. In those individuals confined to seden-

tary lives much can be accomplished by changes of position which allow various viscera to "ptose" away from the localities toward which they conventionally gravitate.

One hears a good deal today about colonic irrigation, which is at times of undoubted value in meeting the obscure toxemias which we are discussing. It is important to bear in mind, however, that the task attempted is usually an Augean one unless the supply of adventitious material is curtailed at the source. In other words, unless the diet be very carefully regulated and reduced the purpose at hand is frustrated, inasmuch as irrigation can rarely keep pace with the introduction of new material from without.

I have just come back from a short trip to Boston, and I want to mention something to you indicative of the fact that the syndrome we are interested in can be brought about in a variety of ways. Dr. Percy Howe, a dentist, has been able to produce dental caries by means other than the presence of bacteria. He has been studying the influence of a diet upon monkeys, rabbits and guinea-pigs, and he has found that it has been perfectly possible to produce dental caries and also arthritis by changes in the diet; and he has reached the viewpoint that the infection of dental caries is a secondary matter. It is known to be by no means as frequent in animals as it is in man, and it was apparently largely absent in the men of the Stone Age. In a prehistoric skull recently found in Rhodesia, and presumably of later date, caries has been observed for the first time.

I leave it to you to consider whether some of the conditions and infections which you meet in the tonsils may be secondary to rather than causative of the syndromes which they accompany.

I think it is of the highest importance in considering this problem to maintain a sane and balanced viewpoint and not to go off at a tangent. We try to hold ourselves down to earth, and it is our desire to advocate no particular panacea or viewpoint. I think we should look at the problem from the broad standpoint of physiology, to which many different causative factors may contribute. You gentlemen are studying one of those factors,

in a useful and important way, and I know you realize that the removal of foci does not cure these individuals in every case. I think it of importance for us all to know when and why it does not work. So if you regard arthritis as only one big branch of a large tree, the other branches being made up of a host of other conditions, and if you realize that a great many conditions can bring this situation about, it seems to me easier to understand what kind of a disturbance we are dealing with and why it is that we fail sometimes to get the results that we want.

I am quite sure I have not told you exactly what you want to know, which is when to take the tonsils out. I cannot do this, but I can say that it is possible at times, by means of the laboratory observations I have mentioned, to determine whether an individual is really suffering from a disturbed physiology in the absence of clear local or clinical evidence to that end. I could cite many illustrations to exemplify this fact and also to show what can be done toward restoring to health persons suffering from one or another form of the syndrome we have been discussing. If you think at all of what I have tried to put before you to night, I beg of you to believe that the danger lies in taking an extreme viewpoint and in regarding this or that measure as specific. The only dependable treatment of these cases consists in regarding them as suffering from a disturbance of physiology which can be induced by many agencies and may require many accessory forms of treatment for its restoration even after the original cause is removed.

DISCUSSION

Dr. George Fetterolf: Dr. Pemberton has shown us one side of the field—the immediate results in those cases in which there was decided improvement for a few days. In some cases we see patients whose symptoms are very much worse the first few days. Does Dr. Pemberton believe that this is due to the extra dose of toxins which is forced into the circulation during tonsillectomy?

My own dentist is quite a crank on the subject of sugar. His belief is that dental caries depends largely on the amount of sugar used. I think he said dental caries is very common in the United States and very uncom-

mon in Italy, and to support his view the figures he gave were these: That in the United States the per capita sugar consumption is about 110 pounds a year and in Italy it is something like 8 or 9 pounds a year. Is the question of sugar important in the question of dental caries?

Dr. George B. Wood: I have just one question. Is it possible, from the clinical study of a case of systemic infection, to determine whether the disturbance in metabolism is due to an infection *per se* or to some other cause, such as exposure or improper diet? Such a differentiation would, of course, help in placing the responsibility on the tonsil, as it is only in the infective cases that we can possibly look upon the tonsil as the source of the trouble.

Dr. James A. Babbitt: I think we owe Dr. Pemberton many thanks for his modest way of showing us so much unplowed ground in this field. I would like to ask the doctor two questions: Just how much does the blood analysis have to do in pointing toward definite infection? How much analogy in this arthritic syndrome is there toward the verifying of osteitis of the internal capsule in otosclerosis?

I am very much interested, in addition to the type of cases that Dr. Eves mentioned, in a number of people who simply seem to present something which is very analogous to the sensation of uneasiness, not pain or fever. I was asked to examine a youngster who was on the point of suicide, he had lost weight and was very much depressed. I looked at his tonsils and thought they must come out, and discovered an old carious tooth-root. A dentist extracted the tooth, obtained a streptococcus organism from it and the youngster immediately recovered. There was a syndrome of general depression. Now, is not that in exactly the same line as the idea of Dr. Pemberton?

Dr. Pemberton (closing): As to Dr. Fetterolf's question, I have heard the same view expressed elsewhere. There is reason to believe that there is something in that statement. I think it was Dr. Eves who said to me that he had seen a case of tonsillitis which, if I recall correctly, was very much improved by the elimination of the use of large amounts of sugar. I am prepared to believe that there is a very real relation tied up in those facts, and the work of Howe, in Boston, shows that the diet does affect the metabolism in remote particulars, such as dental caries.

What we do in our diets is to cut down the total caloric intake, and to do so by cutting down the element which supplies most of our calories in ordinary conditions in life, viz., carbohydrate. In the army about 70 per cent of the food value came from meat and such carbohydrates as

potatoes, bread, rice-pudding, etc. One did not get greens and fruit. The former is the class of food that the poorer classes mostly eat. So if you cut down the carbohydrate element you cut down largely the number of calories. You may perhaps not recall that of the meat eaten nearly 60 per cent goes to carbohydrate. So when a man cuts off meat he also cuts off a lot of carbohydrates and calories at the same time.

As to Dr. Fetterolf's question about getting worse after operation, we can explain that upon two bases: One is that we have given a further chance for the infective organism to become operative. But, furthermore, we also do something else in the way of upsetting further an already unstable equilibrium, and I think that can be illustrated best with the sinus cases. Dr. Stauffer, Dr. Hunter and others of my colleagues have operated on a lot of sinus cases for me and I am getting more conservative as I go on about having these cases operated on. They often get worse instead of better, unless it be a simple case of an infection of the antrum. I think these individuals should often be let alone and treated by accessory measures.

As to Dr. Babbitt's question regarding the leukocyte count and so on, I think a leukocytosis suggests a septic affair, and I rather regard that type of case as one in which there is something of a focally infective nature going on. It is also important to point out that where there is a focal infection, the very fact that it exists paves the way for other factors to become operative. So when you relieve a man's tonsils he may still remain depressed at other places. I want to make very clear my belief that the removal of focal infection does not necessarily complete our duty toward the individual. It is often necessary to institute other measures as well afterward.

As to otosclerosis, since Dr. Hunter has called our attention to this, we have observed a lot of tinnitus among our cases.

As to the suicidal tendency which Dr. Babbitt mentioned, I am glad he brought it up. It illustrates well the general nature of the problem we are discussing. Who is going to be so bold as to say that such a man has organism in his cerebrum? It is infinitely more reasonable and much easier of defense to believe that the individual is suffering from a disturbance of physiology common to the several structures concerned. There is no question that sometimes organisms get into joints, especially in the gonorrheal type; but that they are present in the nerves in neuritis, in the muscles of the head in headache, in the eye or in the skin, as in scleroderma and erythema multiforme, I cannot believe.

Following a talk along somewhat parallel lines which I was asked to give before the Neurological Society recently, one of the members said to me that he was beginning to realize how farsighted Dr. S. Weir Mitchell had been in founding the Orthopedic Hospital when he had included

there nearly all the mechanism for following out the régime which I have spoken of here tonight.

As to vaccines, which Dr. Cohen mentioned, I have not great faith in them. I have not had much success with them myself, and the group of physicians in Boston whom I have mentioned, I think, has given them up. I have seen some cases in which vaccine did help and believe there is often reason to consider them, but I must say I have been disappointed in what they really do bring about.

FEBRUARY 21

Progressive Middle-ear Deafness

BY JAMES BORDLEY, JR., M.D.
BALTIMORE, MD.

I want to thank you for the privilege which you have accorded me, and I want to say that it embarrasses me much to be able to bring to your consideration only the jottings from a few case records. Indeed, I feel that I have only just arrived at a point in my study of deafness which entitled me to ask pertinent questions rather than to be giving suggestions. Still, as you have offered me a splendid opportunity to report certain results which have been noted in the treatment of individuals afflicted by what we are wont to term progressive middle-ear deafness, I accept your invitation both reluctantly and with pleasure.

In the selection of the patients whom I shall here discuss, I have been guided by a desire to eliminate all cases the complications of which might cast a doubt upon the diagnosis, and because I have in mind certain facts to which I wish to call especial attention. Perhaps all of my deductions will not meet with your full approbation, for as Poehlman says, "Facts are one thing, but the interpretation of facts is dependent on the personal equation and the limitations of the observer." My hearers will please bear this in mind, and if I seem to overreach the mark in my conclusions, I hope my very evident limitations will soften their criticism.

The literature of this subject is too voluminous to review, its authors too numerous to fully quote. It is quite probable that in the study of the subject I have absorbed not only the sense but the words of some of these esteemed authorities, so in advance I freely apologize if I quote without credit and beg of the offended authors to weigh their assistance to me as against the offense which I may have offered.

After all we are working for a common cause—the restoration of hearing. And what is hearing? It is the faculty of the perception of sound, or, to quote Helmholtz, "If you admire paradoxes, you may say ærial vibrations do not become sound until. they fall upon a human ear." He implies, of course, an ear in which the physical apparatus permits of physiological function He as the physicist thinks of the normal, we as otologists deal with the pathological. So to us are left those individuals who through disease or accident fall short in their natural requirements. And we must shoulder that burden even though medical tradition has left but little to inspire us. Still, tradition is only hindrance and not a bar to achievement, so in dealing with progressive deafness it might be well to bear in mind the age-old saying, that, "He who looks upon the clouds never sows." Experience has shown us that the proportion of cases that can be cured in the scientific acceptation of that word is indeed small, but those who can be helped I am sure is quite large. By "helped," I do not mean necessarily to imply a marked betterment in hearing, but rather a relief of one or more of the symptoms which have become a constant dread, and by the upbuilding of a morale which will be a bulwark against the mental phase of the affliction. While in my report to you I shall deal only with the physical ear, I have learned with Helmholtz to recognize a spiritual ear.

Progressive middle-ear deafness, as this communication will treat of it, implies an interference in conduction of sound waves through the tympanic cavity. Such deafness depends upon three factors: Vibratory response of the tympanic membrane, changes in the relationship of the bones comprising the ossicular chain and intra-auricular pressure. Through an examination

of the literature, by experiments and from clinical experience, I am led to believe that the tympanic membrane, malleus and incus are essential to perfect but not useful hearing. Indeed, they frequently suffer severe changes without materially interfering with the usefulness of their containing ear. The stapes, however, controlling as it does the stability of intralabyrinthine pressure, is indispensable, because such stability is the very foundation of useful audition. These structures of the middle ear are subject to the influences of atmospheric pressure, and except for the ventilation of the tympanic cavity by the Eustachian tube, would be ceaselessly under greater tension than is normally required for their functions. It can be said that stability of intratympanic pressure is the prime object in the arrangement of the tympanic cavity and its contents, and that upon such stability depends not only the conduction of sound through the middle ear, but likewise through the internal ear. There is little wonder then that in middle-ear deafness we observe defects in perception—of such an association there exists ample pathological and clinical proof—therefore, before we accept any hypothesis for combined deafness, we must weigh well this fact. So far as I am aware, no pathological evidence has been offered which casts a doubt on this mechanical theory for combined deafness, and I feel that the experiments of Dean and Bunch, who with their audiometer found that perception changes result from so simple a lesion as perforation of the tympanic membrane, add emphasis to the thought that physical perfection of the conduction apparatus is essential for perfect perception. Thus reasoning, I am led to the conclusion that changes in the middle ear cannot be dismissed, as has been suggested, as minor factors in the study of progressive deafness. I am willing to have it inferred from this statement that I believe changes in the drum and ossicular chain are provocative of profound disturbances in hearing only when they interfere with normal intralabyrinthine balance. I offer as proof of this conviction a single illustration: A teacher of violin, some years ago, through suppurative otitis, lost the whole of his right membrana vibrans and much of his left. In his right ear he is profoundly

deaf. The remnant of the left drum has become attached by its free margin to the internal wall in such a way that it covers the foramen rotundum but leaves free behind it the tympanic orifice of the Eustachian tube. This individual has a perfect appreciation of timbre and pitch, but has definite tonal gaps for the higher vibrations. By experimenting with him, I found with air in the tympanic cavity under pressure that he had complete restoration of tonal perception without interference either in pitch or in timbre. In my notes on this patient I wrote, "The foramen rotundum closed; the foot-plate of the stirrup pushed in; increased intralabyrinthine pressure." This thought seems justified when, without visible changes other than distention of the tympanic membrane and rotation of the malleus, the hearing to all intents and purposes became normal. Certainly, in this man, by no stretch of the imagination can we associate the tonal loss with poisoning of the perception elements.

If I am correct in my conclusions that normal intralabyrinthine pressure is essential to useful hearing, it will be well for us to consider that agency most concerned in its stabilization—I refer to the Eustachian tube. This tube, in order to carry on its physiological requirements, must open and close sufficiently often to prevent extremes of condensation and rarefaction of air in the tympanic cavity. For many years its permeability has been determined by various methods of forced inflation and obstructed deglutition. Experiments in which I have for several years been engaged have convinced me that as tests for the permeability of the tube these methods are excellent, but as proof that the tube is performing its normal physiological function they are not reliable. They both require force and abnormal action, neither of which exists except under unusual circumstances. It is important in making such observations that we do not disturb prevailing conditions. It has been found that, as a measure of the inflow of air, the ear manometer of Politzer offers much valuable assistance. Politzer proved that air forced through the tube and entering the ear will drive out the drum and thus condense the air in the external canal. A bent tube hermetically sealed into

the external meatus of the canal and carrying a drop of colored fluid will, through changes in the level of the fluid, demonstrate condensation and rarefaction of air in the external and middle ears. I found by experiment on normal ears that forced inflation is not always an essential, but by increasing the caliber of the tube within the meatus and decreasing it at the curved portion containing the fluid by magnification it is often perfectly possible to watch changes in the fluid level even in the ordinary throat reactions during conversation. By substituting a straight tube upon which has been blown a collodion bubble, as suggested to me by Prof. Robert Wood, of the department of physics of the Johns Hopkins University, the slightest alteration in the tension of the drum is made manifest by changes in the iridescence of the bubble. By this simple instrument it was found that in most individuals with normal acuity of hearing that during conversation which, from time to time, requires deglutition there was a change in iridescence; in a vast majority of patients with plastic and hypertrophic otitis there was no such change, and in atrophy of the tube there was most often an exaggerated play of color. It is quite true that in a proportion of patients with chronic middle-ear deafness of the hypertrophic type just as active changes were noted in the iridescence as in normal individuals. But when we think of these exceptions we must recall that hypertrophy frequently gives way to atrophy and that the tests may have been applied during such transitional period; had they been utilized earlier a different result might have been obtained.

Once finding that the tube is not responding to the normal demands upon it, the character of the obstruction and its location should be determined by various types of probes. We will discover by their use that complete loss of permeability of the auditive tube is indeed rare, and I entertain no doubt that in practically all cases of conduction deafness there is, from time to time, ventilation of the tympanic cavity by unintentional forced inflation, and that the longer the elapsed time between these inflations the more profound will become the deafness. When at last Nature opens the tube through atrophy, the damage to both the con-

duction and perception portions of the ear precludes the reëstablishment of normal or near normal hearing. I believe too much stress cannot be placed upon thorough investigation of the Eustachian tube. Results of treatment, as well as of tests, have convinced me that its physiological reactions do not always correspond to its so-called permeability.

Now there are many reasons for tubal obstruction, and most of them are so well known that they do not require repetition. There are one or two possibilities, however, to which I wish to call special attention. Some years ago, while experimenting on the faucial tonsil, I was struck by the capricious action of bacteria, in that a seemingly normal tonsil might be the host for virulent organisms. That these organisms might lie not only in the tonsil crypts but in the stroma itself, without producing visible evidence of damage to the tonsils and still through their dissemination seriously affect remote organs. It was equally clearly demonstrated that the normal immunity of the tonsil stroma could be greatly lessened by changes in its environment—either through gaps in its protective epithelium or through excessive connective-tissue formation within the body of the tonsil.

When I thought of these experiments and associated them in my mind with chronic progressive middle-ear deafness I could but recall the pharyngeal tonsil and the lymphoid tissue in the Eustachian tube. This suggested a possible avenue for the entrance of deafness.

Since that time I have more or less diligently worked to prove the association of deafness and disturbances in the lymphoid elements of the tube. So far I have discovered only what other observers before me have found, that lymphoid tissue is a normal constitutent of the tube and that its cells infiltrate the tunica propria of loose connective tissue below the mucous membrane, thus giving rise to a true adenoid structure. Its profusion and distribution vary with age: while it can at times in early childhood be found in every part of the tube, in the adult it is usually present in profusion only in the pharyngeal end. In old age and from disease it may entirely disappear. It seems undoubtedly to

react to such stimulations as catarrhal and infectious processes in the nose and throat and to be subject to hypertrophic and hyperplastic changes from specific fevers as are all lymphoid structures. Situated in its greatest profusion in the lower end of the tube, associated through origin and contiguity with the pharyngeal tonsil, having its afferent ducts emptying in common with those of the nasopharynx into the retropharyngeal gland, drained by the valveless veins of the pharyngeal plexus, it presents a very possible cause for obstruction and infection of the lumen of the tube.

May I digress here to say that while studying the lymphoid structure of the tube a search was made to find any contained organism which might produce progressive deafness. In this respect I was as unfortunate as other observers. The only suggestive result was the finding in the submucous tissue of the tubes of a number of individuals of an unrecognized bacillus which we were never able to find free in the lumen of the tube. This discovery was first made in a deaf man as the result of deep scarification of an Eustachian tube with a specially constructed spring wire. Cultures were taken in duplicate from the lumen of the tube and from the scrapings. From the latter there developed on fresh tissue by anaërobic methods, bacilli which when placed in the conjunctival sac were harmless, but when injected into the subconjunctival tissue set up a slowly advancing episcleritis, under the periosteum a periostitis with bone softening. Experiments have failed to connect this organism with progressive deafness, and it is mentioned only because it has been suggested that in each individual suffering from deafness there is a specific organism the toxin of which is selective for certain of the perceptive elements, and as such a possibility it is at least interesting.

Now to return to the causes of tubal closure. Emerson and others have pointed out two possible factors in tubal obstruction and deafness upon which I feel too great stress cannot be placed, namely, adenoid masses in the fossæ of Rosenmüller and adhesive bands which stretch between the pharyngeal end of the tube and the walls of the nasopharynx. Emerson points out the fact that

frequently only by digital examination can their presence be determined. Out of 60 individuals with normal hearing, these structures were discovered in but 4, while in 186 out of 300 patients with conduction deafness they were present. The adenoid masses in the fossæ of Rosenmüller are probably misplaced portions of the faucial tonsil and are subject to all of the derangements of lymphoid tissues, and should, in my judgment, always be eradicated. The origin of the adhesive bands is not so clear. In a majority of instances they are probably secondary to disease or injury of the walls of the nasopharynx and the pharyngeal end of the auditive tube. Osteopaths claim that manipulation of the ends of the auditive tubes results in improved hearing. I doubt not that when improved hearing results from such manipulation, it is solely because they have destroyed these adventitious structures, the nature and action of which they little appreciated. The rapid and marked betterment in hearing which oftentimes follows such massage is indeed astonishing.

Difficulties in diagnosis are not limited to our less learned brother, but fall with great weight upon us all, and until we have a more definite knowledge of the ways and means of audition, they will continue stumbling-blocks in the path to prognosis and treatment. We possess no real instruments of precision, so in many cases the otologist must rely upon his accumulated experience. I believe, however, we are at the dawn of a new era in diagnosis, and as instance of that belief, I point with marked appreciation to the highly scientific contributions of Dean and Bunch relating to tonal perception as measured by their audiometer, Sonnenschein's work with Schaefer's resonators, Donal McKenzie's contribution on *Relative Sensitivity of the Ear*, as demonstrated by the alternating phonometer, and Harvey Fletcher's work on *The Nature of Speech and its Interpretation*. Such work makes one feel confident that in the near future we will be able accurately to measure the acuity of hearing.

It has been said that "all hearing tests are based upon negatives." This is quite true in a sense, but reasonably so because our examination is intended to measure the difference between

normal and our patient's loss. As none of the usual tests are free from faults, our conclusions must be based more on the sum of the results than upon any single factor. As Harvey Fletcher says: "Each individual has a hearing characteristic which is quite different from other individuals. . . . Consequently, any distortion of speech will affect some persons differently from others. It is evident then that in discussing speech and hearing we must deal with statistical averages." To this let me add that no otologist can reckon from the averages of other otologists in the measurement of deafness unless he is able to duplicate the methods and conditions upon which those averages were computed. Each observer must establish his own normal from averages personally ascertained, and it is upon such a basis that deviations are calculated in my own work. The methods employed in diagnosis in this series were essentially those in ordinary use and require no elaboration here.

Randall tells us that: "Embryology shows that the Eustachian tube, tympanum and mastoid cells are one . . . structure . . . and that they are portions of the upper air passages as much as the accessory nasal cavities and are lined with an extension of the same . . . mucous membrane with all of its pathological attributes." We must infer from this statement that in diseases of the middle ear we should frequently expect to find changes in the mastoid cells; that such is a fact is clearly revealed by radiographic examination. To quote J. C. Keller: "I consider roentgenography one of the most valuable single aids in the study of the mastoid in health and disease." So, too, have we come to regard it. Dr. F. H. Baetjer, who is in charge of roentgenography at the Johns Hopkins Hospital, has for many months obtained for me such information as a routine on all of my patients. He has adopted a plan through which a comparative study of plates taken at different times will fairly well indicate the progress of the underlying disease. I am sorry that he cannot be here to discuss this phase of the work, because he is, and I am not, qualified to answer questions relating to technical details. It must suffice for me to show some slides obtained during various

stages of chronic otitis media, and to say that radiograms, like other diagnostic means, must be measured not singly but in association with other tests. Such association is made necessary by the varying types of mastoids and their likelihood to cast confusing shadows, and because the investigator may mistake as new processes shadows left by prior attacks of an acute nature within the mastoid.

In progressive conduction deafness uncomplicated by previous attacks of otitis suppurativa, three changes in the mastoid can be demonstrated: Either separately or together; a clouding of the cells; cell destruction, and sclerosis. I believe it will be impossible to determine whether this sequence always obtains until a large series of cases has been followed from the first to the last stages of progressive conduction deafness, and it is unfortunate that such opportunities are rare. We have observed cell-clouding as an early manifestation, cell-destruction with moderate loss of hearing and sclerosis in the terminal stages of atrophic otitis. The mastoids of many elderly people present evidences of sclerosis. Whether this is a normal change I am not prepared to say, but it is an interesting fact that in several patients of advanced age, but without these mastoid manifestations, the results of treatment were very gratifying. In the rapidly progressive deafness of childhood there is a hazy cloud throughout the mastoid coincident with the appearance of deafness, while in uncomplicated otosclerosis we have found no striking radiographic change.

The portion of the mastoid usually showing changes is that lying within the triangle, or what we might call the antrum portion, and the disease here apparently spreads from the middle ear. Sclerotic changes at first involve the antrum portion and only very gradually include the large pneumatic cells of the inferior posterior portion of the vertical division of the mastoid. Following such involvement there is usually marked shrinkage of the whole mastoid process and profound deafness. Our observations lead us to believe that advancing mastoid changes are closely associated with progressing deafness and that sclerosis of the

mastoid is an exceedingly bad prognostic sign when associated with marked reduction in air-conduction time.

Every patient in this series has, where deemed advisable, been subjected to rigid physical examination by qualified internists to ascertain the possible presence of syphilis, tuberculosis or disturbances in the endocrine glands. Great care has also been devoted to ascertaining probable inherited predisposition, and quite a few family trees have sprung therefrom.

This brings me to the crux of this communication: the treatment of progressive middle-ear deafness. Such treatment "depends upon the local condition found on physical examination in connection with the information obtained by a careful functional examination. . . and it must be borne in mind that the general condition influences the progress of any local inflammation." This is the summation of the question of treatment made by that vastly experienced American otologist, Dr. Dench. Treatment which does not conform to these requirements certainly will not be productive of lasting benefit. During my professional career I have run the gamut of all forms of treatment from the hot onion to ossiculectomy. In that time I have seen many devices of great ingenuity contrived, many theories blasted and many dreams destroyed. Still these attempts were but milestones in our advancement and played their part even though many are now despised and discarded. If they taught us no more than the futility of trying to restore function by attacking the results rather than the cause, we have benefited by their exploitation.

I have been guided in my efforts to assist the deaf less by my own ingenuity than by knowledge acquired of the successes and failures of my predecessors and contemporaries. I have no specific to offer for the cure of deafness nor do I opine that I ever shall have. I am here simply to direct your attention to some purely arbitrary methods of attack and the results gained thereby. Every patient is treated with the idea that betterment will follow only persistence and patience, and they usually reap my expectations.

I have eliminated from this list all patients with syphilis, tuberculosis, primary perception deafness and coexisting suppurative otitis media. The reason for this is quite obvious. I wish to see the results of one line of treatment uncomplicated by other factors. Treatment to be curative would have to restore normal functional responses in both the middle and internal ears. This is infrequently possible. To be helpful it must restore the ability to indulge in ordinary conversation without effort or strain. This is frequently possible.

All of the patients in this series were treated by one plan: The Eustachian tube was freed of all adhesive bands; the fossæ of Rosenmüller cleared of all adenoid masses; the nasal mucosa kept as free as possible of swelling and the nasopharynx from excessive mucous secretions. Where indicated, the general physical condition received attention. With the wire devised by Yankauer the auditive tube was dilated and treated, first, with a solution of nitrate of silver followed by iodine solutions, mercurochrome or meroxyl[1] (the reason for such probings was a desire first to destroy all possible granulations); second, to destroy the surface epithelium and its contained organisms; third, to make more patulous the lumen of the tube by destroying organic strictures. It was believed wise to keep the tube as sterile as possible for a period of two weeks or longer in order to make permanent any gain in permeability. The cotton-tipped wire was forced not only through the tube but into the middle ear. This was found necessary because at the tympanic entrance to the tube there are frequently to be encountered adhesive bands which block the inflow of air. By bending the end of the wire it has been found entirely possible to pass it into practically any part of the tympanic cavity. Many adhesions between the drum and internal wall and the handle of the malleus and internal wall were thus stretched and frequently destroyed. Marked changes in circulation resulted from this mechanicochemical stimula-

[1] Through the courtesy of Hugh H. Young, M.D., director of the Brady Institute, Johns Hopkins Hospital, I have had furnished me from their laboratory the meroxyl used in my investigations.

tion of the tympanic cavity, thus favoring the absorption of inflammatory deposits. A not infrequent obstruction to the inflow of air into the tube is the adherence of the drum to the wall of the tympanic cavity at or near the tubal entrance. The passage of some form of probe, preferably the wire, was essential if it were to be freed. As a rule, the adherent portion of the drum was found much stretched when freed; a linear scar, produced by an incision through its long diameter, will cause it to function better. The dilatation of the tube with the wire was found to be more readily effected when a 3 per cent solution of nitrate of silver was used for 3 applications, followed by 12 applications of a 2 per cent solution of meroxyl. Upon returning after the initial series of treatments, meroxyl alone was used without evidence of unsatisfactory dilatation was found, then nitrate of silver was used at the first visit and meroxyl subsequently. Lapses of six weeks, with seances of three days each for treatment, were continued for at least one year, without indications in the ear or in the state of the hearing made it seem advisable either to increase the frequency of the visits or to discontinue treatment altogether. The passage of wires is attended by pain which varies with the individual. It can be lessened by forcing solutions of cocaine into the auditive tube. The too constant use, however, of cocaine is to be avoided because it softens the epithelium and produces a congestion of the tube. Where nitrate of silver is to be passed, cocaine is necessary, and for this purpose a 2 per cent solution seems best. I have found it a wise precaution to discontinue all probings during mild attacks of myringitis which are occasionally met with and which last for about forty-eight hours, and are readily controlled by either a solution of carbolized glycerine or an ice-bag over the mastoid. Acute tubal closure sometimes results from the passage of the wire during acute inflammation of the nose and throat. As such attacks are provocative of trouble, I now refrain from dilating the tube at such times.

In this series of 300 patients, wires were passed over 10,000 times. The only serious consequences were 6 instances of acute suppurative otitis in patients who had some time prior to treat-

ment suffered from similar infections; 2 patients had excessive dilatation of their tubes and further impairment of hearing (these latter were among the earliest patients so treated); and when more strenuous efforts were made for rapid dilatation a few suffered transitory vertigenous attacks at the time of the passage of the wire; in 2 such patients these attacks assumed the proportions and symptoms of a mild Menière syndrome and lasted in one of them two and in the other nine days. All patients were cautioned to avoid physical and nervous excesses; sea- and river-bathing; to abstain from excessive use of alcohol and tobacco; never to take quinine or the salicylates. They were provided with a solution of aconite to be taken as a preventive or cure for choryza; a mild iodine gargle where indicated; a menthol-eucalyptus solution for daily use as a nasal spray. In addition, they were instructed in what we call "Randall's ear massage"—gentle suction and pressure with the palm of the hand over the external meatus, this to be practised twice a day. Exercises designed to develop existing hearing were always prescribed.

Briefly, all patients in this group had been deaf for not less than two years and were selected from among nearly 1000 cases, because of the assurance given each of them, either by some other otologist or by myself, that treatment could prove of no avail. In every case the treatment was kept up for at least one year. A report on the results of their treatment seem particularly desirable because the final record was made not less than one year subsequent to the discontinuance of regular treatment; no case is recorded as improved without there was an increase in acuity for the voice, a lowering of the lower tonal limits and an increase in air-conduction time.

A number of interesting facts developed from a statistical review of these 300 patients. In the first place it was found that age in itself was no bar to improvement. That a majority of those benefited were past forty-five years of age; the class least benefited was composed of those under twenty. In the older people I surmise that one deals only with the results of past infections and inflammations rather than with an active process. If the patient has preserved a modicum of hearing through the

active period, I am inclined to look with favor upon treatment, provided signs of atrophy of the drum and Eustachian tube are not present and the mastoid is clear. There were found four general classes into which it was possible to place all patients. This classification is rather arbitrary and is equally based upon symptoms and results of treatment. The first of these, simple atrophy, is probably but the terminal stage of a preceding hypertrophy. It is characterized by a too patulous tube, atrophy of the drum and sclerosis of the mastoid associated with loss in air and bone-conduction time, constriction of both ends of the tonal limits and marked loss of voice acuity.

Into the second group were placed the simple otosclerotics. Those with no changes in the tympanic membrane, auditive tube or the mastoid, and with loss for air, conduction time, an increase for bone-conduction time and an elevation of the lower tone limit associated with gradual loss in voice acuity.

The third group, which we will call acute combined deafness, was composed entirely of children from seven to sixteen years, who, following some acute infection, rapidly became profoundly deaf. The first signs were typically those of middle-ear deafness—a tightly closed Eustachian tube, sharply indrawn drum, loss for the lower forks and in air-conduction time. In a matter of weeks a pronounced involvement of the labyrinth manifested itself. A majority of these little patients became primarily deaf either during or immediately following pneumonia. All of the patients in the fourth or last group are classed as simple combined deafness. They do not all present the symptoms common to uncomplicated middle-ear deafness. Some, indeed, from the standpoint of symptomatology, might readily be put in the otosclerotic group. Not a few came to me with such a diagnosis, or I so classed them. Still, it is impossible to believe that treatment such as was used could have cleared up otosclerotic bone changes or primary labyrinthine involvement. While they reacted to tests as did other patients classed in the former group, they did not react to treatment; therefore, it was thought expedient to look upon the diagnosis as incorrect and call them cases of simple combined deafness.

TABLE I.—CLASSIFICATION AND RESULTS OF TREATMENT OF 300 CASES

	Atrophy.	Otosclerosis.	Acute combined.	Combined.
No. of cases	55	6	12	227
Improved	None	None	None	8 normal 5 normal (one ear) 18 (50 per cent) 21 useful 26 by tests
Unimproved	55	6	12	149

TABLE II.—FIRST EXAMINATION. SHOWING TESTS AT ORIGINAL VISIT OF 4 PATIENTS

Age	56	42	34	23
Duration	18	20	6	13
Family	4	0	18	1
Voice R. E.	3	8	1	7
Voice L. E.	9	4	10	7
B. C. R. E.	60	55	40	65
B. C. L. E.	60	60	65	65
A. C. R. E.	10	5	30	10
A. C. L. E.	15	20	20	15
Treatment (months)	16	12	14	18

TABLE III.—LAST EXAMINATION. SAME PATIENTS ILLUSTRATED IN TABLE II. RESULT OF ONE YEAR'S TREATMENT

Age	56	42	34	23
Voice R. E.	25	25	1	25
Voice L. E.	9	15	25	25
B. C. R. E.	60	55	35	65
B. C. L. E.	60	60	65	65
A. C. R. E.	55	70	20	80
A. C. L. E.	20	50	70	20

TABLE IV.—FIFTY PER CENT HEARING. SHOWING RESULTS OF TREATMENT IN THE MORE IMPROVED EAR OF ANOTHER GROUP OF PATIENTS

Age	69	54	46	45	43	36
Duration	20	16	4	16	11	8
Family	2	0	0	0	8	0
Voice R. E.	3	2	7	6	7	11
Voice L. E.	12	15	15	14	16	20
B. C. R. E.	60	55	60	35	60	60
B. C. L. E.	60	50	60	60	55	60
A. C. R. E.	5	10	30	25	10	15
A. C. L. E.	50	80	60	65	60	75

TABLE V.—FIRST EXAMINATION. SHOWING PRIMARY TESTS IN GROUP OF BILATERAL RECOVERY. VOICE TEST AVERAGE FOR BOTH EARS

Age.		75	74	64	61	56	37	26	18
Duration		21	27	12	8	20	3	2	2
Family		0	12	?	0	5	0	1	1
Voice		3	15	20	8	20	2	4	10
B. C.	R. E.	45	40	50	35	50	70	65	45
	L. E.	55	40	40	60	50	65	50	45
A. C.	R. E.	12	40	35	6	30	10	3	20
	L. E.	20	35	45	12	30	15	17	22
Treatment (months)		14	21	12	16	16	12	18	12

TABLE VI.—LAST EXAMINATION. SAME GROUP SHOWN IN TABLE V. RESULTS OF TREATMENT

Age.		75	74	64	61	56	37	26	18
Voice	R. E.	3	15	20	8	20	2	4	10
	L. E.	25	25	25	25	25	25	25	25
B. C.	R. E.	50	40	45	45	50	65	55	45
	L. E.	50	40	45	45	60	65	60	60
A. C.	R. E.	16	35	35	9	30	12	10	21
	L. E.	60	55	60	55	70	65	60	65

I will show with the lantern the results of treatment in the various groups; but before I proceed to that let me briefly state what the pictures show: In the atrophic group 55 patients were placed. None of the patients in this group showed the slightest benefit from treatment. In the simple otosclerotic group there were 6 patients, none of whom improved; in the acute combined group we find 12 children, all of whom failed to respond; in the simple combined there were 227 patients: of these 8 recovered normal hearing in both ears; 5 in one ear; 18 hearing of better than 50 per cent in one or both ears; 21 regained sufficient hearing to make the ordinary duties of life possible and 26 additional showed improvement by tests, but to an insufficient degree to be appreciated by the patients.

It is interesting to note that where patients make substantial gains they do not relapse. In but 2 instances have I seen such advancement lost; in fact, the proportion of patients who have gained, even months after regular treatment had been discontinued, is far more impressive. And this hope I always confess is kept tucked back in my mind.

Among those who are counted as failures there is a very definite if small class who are convinced that something unexplainable has taken place as the result of treatment. It is variously called relief from mental oppression and relief from nervous tension. Some of these patients have given me more solid satisfaction than almost any others. It matters little whether such relief found its origin in a changed mental attitude or not, so long as it brought happiness and contentment. Deafness is only a handicap, and if we succeed in helping to relieve its burden, our work has not been in vain, for after all it is the duty of the physician not only to heal the sick but likewise to comfort the afflicted.

DISCUSSION

Dr. B. Alexander Randall: I must admit that I am rather appalled at the idea of discussing the many topics which have been so ably presented, and I am also still more appalled at the large gaps in my own knowledge of a great deal that has been brought forward, and feel in a very chastened mood as I have listened to what has been brought forward, and am a good deal at a loss as to what best to take up.

One of the points that I would like to speak of in passing, at least, is with regard to the bands in Rosenmüller's fossæ, because in one of the earler meetings of this Society, some thirty odd years ago, Harrison Allen told me about the importance of these bands; and since that time Myles, of New York, has talked on the subject, and Jervey, of North Carolina, also referred to it, probably ten years or so ago; and since that time I have only noticed the osteopaths saying anything in regard to it. It is, of course, very interesting, but I think they are hardly as far in relation to priority as has been suggested, unless the Doctor knows of more instances than have come to my knowledge in regard to the breaking up of these bands and the presence of adenoid processes either in the mouth or in Rosenmüller's fossæ.

The traumatism in regard to these things I had not known of, but regard them as being the atrophic remains of previous adenoid tissue undergoing changes. I fail to admit the osteopathic discovery of those matters.

The matter of hearing tests has always interested me, and the matter of the voice test seems to me one of very great importance, not always fully appreciated and perhaps not fully brought out by Dr. Bordley in his talk. To my thinking the loss of the high tones is sometimes merely neurasthenic and temporary. The patient can hear every note of instrumental music perhaps, but within their range may have perhaps a very fair amount of hearing, except for these extremely subtle consonant sounds. The sounds may be very easily recognized, but their perception of the voice as a means of communication is very seriously damaged, and this I think is in relation to the impairment of the high tones, which is a matter of nerve impairment in most cases, sometimes permanent, but in most cases purely neurasthenic and ready to yield to a little treatment with strychnia and so on.

Regarding the point of the teeth-marked tongue, I do not find any reference to this in books, but to me it seems to be of very material importance, as indicating the patient who, with a flabby tongue, has the flabby general condition of the nervous system.

I was interested in passing as to the reference of Dean's otometer, and would like to ask Dr. Bordley how far he accepts its readings. To my mind the otometer, like the acousticon and other forms of telephonic apparatus, simply transposes the sound waves into a pitch so different from what they often purport to be that I cannot accept the readings of those things as meaning all that is claimed for them. I think almost every case of middle-ear deafness hears pretty well over the telephone, perhaps better than other people, because they are not disturbed by other noises.

The roentgenological study is of much interest also, particularly from its clinical aspect. The clinical use of the x-ray, to me, however, has been very slight, and it has, to my thinking, rather blundered, or I blundered upon the value of its use in clinical cases. Bigelow of Providence, has got some excellent results.

The matter of the Eustachian tube is of enormous importance, and our treatment by it is unquestionably the method by which we are to reach a great many of these cases.

In regard to the bougie, I have seen Pollitzer use the bougie in Vienna. Sometimes, in these subacute cases where the fluid was visible in the tympanic cavity, and where the Eustachian stenosis was otherwise uncomfortable, Pollitzer would steep his catgut in silver solution and then pass

it and leave it in a certain length of time in the Eustachian tube. I came back with a strong enthusiasm toward the bougie, tempered with the fact that I have seen some cases where there was only the thinnest mucous membrane between, and wondering how soon I would get through that into the carotid artery and have fatal bleeding. I have had only one case of bleeding, but I lost some of my enthusiasm as I went along, and of late years have made almost no use of the bougie, because with the use of dionin I have accomplished everything desired. But from what Dr. Bordley has told us, it sends me back more toward the employment of the bougie.

I have yet to see the limited number of otosclerosis cases which have come to his notice. I think, after thirty odd years of study I can still number the cases that I have seen on my 10 fingers—I mean by that the cases that are not sclerotic at all. The trees of inheritance have seemed to me to grow in very thin soil. I should say they thrive in hot air.

As to Yankauer probes, I have not particularly used them, and it may be by trying them I should find them of value.

As to one point, I am wondering at hearing nothing at all in regard to the tuning-fork test over the nostrils. Thirty years ago Pollitzer put forward this test, and to me it is a test of extreme value and importance. Where we do not get this we have a very positive Eustachian stenosis.

So much has been put forward that in the time which I am allowed to take it is impossible for me to express at all my appreciation of much that Dr. Bordley has brought forward; but I wish to make these notes and assure him that I will try to reform my ways and feel better for it.

Dr. S. MacCuen Smith: Certainly, the questions brought up by Dr. Bordley are most important, and I, like Dr. Randall, must confess my ignorance in not being very well informed on some of the points that he has brought out.

The function of the Eustachian tube, especially the lymphoid tissue in this tube and the proper treatment of that lymphoid tissue, is of course very important. The two points that are so necessary for good functional activity of the organ of hearing, and especially in these hypertrophic types of disease which the doctor principally treated, are, in the first place, you must have good nasal respiration, and, in the second place, what we may call good tubular respiration. It is absolutely necessary to have this air enter the tube in a fairly physiological way that you may have good functional activity in the organ of hearing. I am speaking particular of the first part of the organ of hearing.

I, like Dr. Randall, would like to just say a word about Rosenmüller's

fossæ and the breaking up of adhesions, etc. I remember very well, in my earlier days, of Dr. Harrison Allen having first called attention to this fact, and during my professional life I always paid a good bit of attention to the breaking up of adhesions, and I have seen some cases in which tonsils and adenoids have been removed for the relief of deafness and the adhesions still remained; but when they were broken up afterward and better action of the tube resulted, the hearing returned, the same as has been related by Dr. Bordley.

In the importance of the *x*-ray, I quite agree with Dr. Bordley that the *x*-ray is important in showing certain conditions not only in the mastoid but perhaps in the middle ear itself. However, I believe it is necessary that we should have an expert to be the interpreter of the *x*-ray. Personally, I never undertake to point out the *x*-ray findings, because those of us in the larger centers of population, at least, all have experts for this interpretation, and if there is any technical part of *x*-ray work, it is the interpretation of the plate. I have had any one of three men particularly in Philadelphia, and I could mention them—Dr. Pancoast, Dr. Pfahler and Dr. Manges—who I think are decidedly expert in this line of work, especially Dr. Manges, who has done most of the work for me, and also Dr. Pfahler who has pointed out these sclerotic changes which have been mentioned by Dr. Bordley.

Regarding the Yankauer method, brought out some years ago, I here must confess that my success was so small and amounted to so little that I almost wholly abandoned this line of treatment, and I am very glad, indeed, that Dr. Bordley has brought the matter before us tonight and has told us how to use it a little more successfully than I have ever been able to obtain, and I feel that I shall take it upon myself to resort to this line of treatment in the future.

Dr. Bordley spoke of certain gymnastics, etc., and I just wondered whether in carrying out these gymnastics it was customary in his work or whether he recommened the use of the neck gymnastics in some of his cases. It has been something that I have been doing for a good many years, and I feel that it is very important. The neck gymnastics simply consist in working your neck in various directions, forward and backward, and avoiding particularly the making of the muscles of the neck rigid from one point to another. It cannot be done when you are dressed up and have a collar on; it should be done at night and in the morning, when going to bed and when rising. It should be done by putting the head as far forward as possible and then back as far as possible, and then from side to side. I am quite sure that has helped some of my cases very much.

I was very much interested in the manner in which Dr. Bordley was

able to apparently break up adhesions of the tympanic cavity by the introduction of these wires, and we can quite conceive that having been able to do that he certainly would get benefit in the great number of cases.

I was very much pleased to see the sharp line that Dr. Bordley drew between the atrophic cases and the hypertrophic cases. I came here with the thought in my mind that perhaps he would be able to throw some light on these atrophic cases, and particularly on that type of cases which in my experience are very rare—the otosclerotic cases. In these atrophic cases, when they are advanced, I have never personally seen any benefit derived. Dr. Bordley showed on the lantern slide something that I took to be a case of hydrops ex vacuo. It has been my habit in those cases to make an incision, do a myringotomy and let out the fluid, and then later I have been able to get those cases open by means of a catheter and bougie.

In regard to the otometer, it has been my pleasure to see Dr. Dean, in New York, and the question of this otometer was brought up, and my impression is that they are not using it very much any more, but it is being used for scientific purposes by his copartner.

Dr. Hays (New York): I think Dr. Bordley has been extremely modest in his statements this evening, particularly if I think of the statements which I have heard from a large number of his patients in New York. I have been anxious to hear from him to see whether it were possible that he was doing something that was really new and which I feel we could accomplish a great deal by using. It would be almost impossible to take up in detail all the subjects that Dr. Bordley has spoken about, but I think two of them are very important. One is the inflammatory condition around the Eustachian tube, which by no means is due in all cases to adhesions of Rosenmüller's fossæ, but frequently is due to the tips of the inferior turbinates impingeing on the tubes. We have tried all kinds of methods for reducing them and find that we have two things to be considered: One is the general condition of the patient and the other is some condition in the nose or sinuses. During the last few years we have had high frequency vacuum electrodes made, which we passed into the nose and up to the Eustachian tube and applied for five or ten minutes, with more or less good results in some cases. For the last two years I have attempted to have a nasopharyngoscope made in such a way that I could pass it directly to these growths and bring them off. I do not think we break down adhesions in the fossæ of Rosenmüller as often as we might. I do not think the majority of otologists use their nasopharyngoscopes as often as they should.

For more than ten years we have been using the Yankauer method of dilatation of the Eustachian tube. We have used some instruments similar to the ones Dr. Bordley described, and I wish my tests could show as good results as his. I feel we are right in dilating the tube, but I feel we have not gone far enough. About four years ago I started to do some experimental work and published a paper on it under the title of "Exploratory Tympanotomy," in which I made an incision behind the ear and through the drum, and with various instruments was able to cut adhesions which bound down the ossicles and the drum. The work was in its experimental stage when war broke out and prevented our going on with it. I believe it did no harm. This brings up one last point which is of a great deal of importance. I think the majority of us have had our office hours disturbed a number of times by agents who have advocated various mechanical contrivances for massaging the middle ear. I have no doubt that automatic massage does some good, but if you use that in these cases your are simply exercising an influence upon the ear drum and you are not getting rid of the causative factor which produces the deafness. It is a question in my mind whether you really get any result from it or not. I believe that instructing your patient to use a certain amount of exercise of the ears and of the neck accomplishes a great deal. We have a printed form of exercises which every one of our patients get, this shows them how to use the muscles of the ear and the neck, and I believe it accomplishes a great deal.

Dr. George B. Wood: I would like to ask Dr. Bordley if when he breaks up the adhesions with a wire it is accompanied by evidence of trauma such as hemorrhage or having trouble with the tympanic cavity filling up with fluid? I do not quite understand how he gets the bent wire into the tympanic cavity. Does it scrape the membrane as it passes through the tube?

Dr. Bordley: It does not hurt it at all.

Dr. Wood: I once tried to open up the Eustachian tube in a dog and I found I was not able to do so. I cut up the mucous membrane around the mouth and the palate.

Dr. Bordley: We had about twelve hemorrhages. They did not amount to anything.

Dr. Nathan P. Stauffer: I found Dr. Bordley's method of *x*-ray examination very beneficial in outlining lateral sinuses which had been

displaced. The x-ray disclosed the sinus and it was not very hard to determine the x-ray interpretation because the usual cells were entirely missing and you could tell the difference between the one side and the other. It is much better in taking those pictures if the man takes first one side and then the other.

In regard to the sclerotic changes, in a number of cases I was impressed with the concrete sclerotic changes, and I thought that might cause the long delay or the less chance of danger complications, because of the sclerotic changes holding off the advance of the change of the germ or possibly its being caused by the long necrotic change going on into the mastoid bone.

In regard to the osteopathic treatment, I have had about a half-dozen cases who have had their noses scraped out by the osteopaths, and I guess they were about as bad afterward as they were before.

Dr. Francis R. Packard: I would like to ask Dr. Bordley whether he uses massage in these cases, and if so, what kind of massage?

Dr. Bordley: I use only the hand massage, with the palm of the hand against the ear.

Dr. R. J. Hunter: I think Dr. Bordley is very modest in showing us that out of 300 cases he has actually only got results from 50 per cent. I think that is accomplishing very much, and particularly do I know that Dr. Bordley is getting to the stage now where the most difficult cases of deafness go to him after they are tried by many of us. I had felt, from a little correspondence that I had with him a year and a half ago, that he was going to tell us of some specific medication that he was using which accomplished these results. I should like to ask Dr. Bordley his inception of the pathology that occurs in the middle ear which causes these adhesions which he breaks up. He spoke of examining his patients for tuberculosis or syphilis. One point that has been brought to my attention in examining a number of cases of arthritis is the frequency with which we find changes in the middle ear and often in the inner ear also in cases of chronic arthritis. Now, these changes were very frequent, and my attention was called to the fact that very often the patients had not noticed any impairment of hearing at all, although we found marked changes in the drum, calcareous deposits in the drum and in some cases spots of atrophy. An important finding in these cases was the frequency with which these patients had tinnitus and vertigo, and the point I think is this, that we must not look at these ear cases from the standpoint of the ear alone. We must examine them from a general constitutional standpoint, because

very frequently in these arthritis cases other ailments possibly had overshadowed their ear condition, or at any rate they had not noticed them; and I think if we emphasized the importance of paying attention to tinnitus and vertigo, we would frequently have picked these cases up before the hearing had been impaired.

Dr. Benjamin H. Shuster: I would like to ask a question or two. First of all, has Dr. Bordley noted the effect on the patient of the tinnitus which is so often associated with this question of deafness about which patients complain as much as about their hearing? And the other question is in regard to the type of wire which he uses. In my associations with Dr. Coates, who is an advocate of the Yankauer method, we have got some good results by this method. The wires that Dr. Yankauer used are rather flimsy things. It seems if a wire would stay kinked it is quite possible for it to break off and remain in the Eustachian tube. Some doctors use a wire like a piano wire. As to the Yankauer wire, if you do bend it and it does not break, I do not see how it will have sufficient strength to break down certain adhesions. Another thing is, How does he get to the roof of the attic? It seems that is where most of the damage is caused.

Dr. George Fetterolf: In the earlier days of my ear work, the Duel method of electrolysis was quite in vogue at one time. Does Dr. Bordley find after ear adhesions are broken up that they recur or do they stay broken up?

Dr. Bordley: No. You have to keep them under treatment for a year at least.

I did not intend to give the osteopath the full credit for the discovery of the cause or the treatment of adhesions and of adenoids in the fossæ of Rosenmüller. I gladly give it to the College of Physicians of Philadelphia.

I did not exactly understand Dr. Hunter's question about the nature of the pathology of the changes in the ear. Do you mean the cause?

Dr. Hunter: Yes. What causes the adhesions?

Dr. Bordley: I would like to be able to answer that. I do not know. I know the general condition of the middle ear when the conditions have formed, but I do not know the cause. I think for every middle-ear trouble there is an infection. Whether that infection lies in the tube, or in the mucous membrane of the middle ear, or in the drum, or in the nose, I do not know, but I think the basis of the whole thing is to be found in some infection somewhere.

Now as to the type of wire used. I will say for the wires used by Dr. Yankauer, you cannot break them. It is next to impossible. I have never succeeded in breaking them yet. The wire that I put into the middle ear is a wire the end of which has been passed through a flame. I can get the bend on the wire that I want. I can get more resistance from that than from the ordinary wire.

I started with a small catheter that is closely applied to the Eustachian tube and I have seen no damage done. I have seen a few hemorrhages in the middle ear, but they have done no damage. I cannot see any harm resulting from it. Of course, unfortunately, there were not 50 per cent of the patients that I reported who were much better. I think it was nearer 30 per cent.

I think that of the actual changes, so far as the practicability of the hearing was concerned, it was about 10 per cent.

MARCH 21

Report of a Case of a Misplaced Middle Turbinate (with Exhibition of Specimen)

BY GEORGE B. WOOD, M.D.

This patient is a young man, aged about thirty years, who came into my office complaining of obstruction to nasal breathing. The front part of his nose was perfectly normal. The septum was slightly deflected to the left. There appeared to be a marked enlargement of the posterior and of the left inferior turbinate. It was typical in appearance and position, but possibly a little too high up and too far back. After cocainizing his nose and looking in anteriorly, this growth did not seem to be attached to the inferior turbinate. Then I had a suspicion that it might be some form of malignant tumor springing from the lateral pharyngeal wall just above the Eustachian tube. I used a cautery snare for its removal. As I got part way through the growth the patient moved his head suddenly and the thing came away. On attempting to pull it out I had difficulty, but I finally did worm it out at the front of his nose. Then I looked in his nose and I found that

his inferior turbinate was entirely intact. Some five or six years previously he had had the greater portion of his middle turbinate removed and some of the anterior ethmoidal cells opened up. The hemorrhage was so severe at that time that it needed packing immediately. When I examined the specimen that I had removed, I found that the only point of attachment was at the extreme posterior end, and that it represented practically the whole of his middle turbinate, which apparently had been cut off and had been pushed back into his nasopharynx with the anterior end hanging down behind the posterior end of his inferior turbinate. The end hanging down had become hypertrophied, so that it resembled a hypertrophy of the posterior end of the inferior turbinate.

Report of a Case of Microtia, with Absence of the External Auditory Canal

PRESENTED BY NATHAN P. STAUFFER, M.D.
READ BY ORAM R. KLINE, M.D.

E. S., a female infant, aged four months. The eleventh child of healthy parents. Eight of the children are living and well. One died in infancy—"a blue baby." The other died, at the age of six months, with pneumonia.

The mother gives a history of three miscarriages, two of which occurred just before the pregnancy, which terminated in the birth of this child at term. Only one of the miscarriages was spontaneous.

The infant was first brought to the out-patient department of the Presbyterian Hospital, of this city, for treatment, December 22, 1922. It presented the following appearance:

An emaciated child, one month of age, weighing 6 pounds, 10 ounces. Eyeballs prominent, even protruding to the point where the upper lids partially turned inside out when the infant cried. Nose completely blocked as far as respiration was con-

cerned, but mucopurulent discharge was forced from both nostrils by forceful crying. Mouth disproportionately large and always open, presenting a large, broad tongue partially tied down by the frenum. Oral respiration only, and this was a combination of gasping, coughing and strangling when irritated in any way.

Ears. The pinna on either side, only a tab of tissue practically devoid of cartilage, pushed forward and adherent along its anterior border, bearing very little resemblance to that of the normal ear. In these tabs are found several dimples, but all are shallow and end blindly, no true external auditory canal being found.

There is very little change in the appearance of the child at this date, three months later, except that respiration is less labored. It has gained only 10½ ounces in weight.

Dr. Newcomet has made an *x*-ray study of the case and has reported the presence of small osseous external auditory canals. From this report we assume that the atresia is only membranous.

It is our intention at an early date to attempt to locate the orifices of these bony canals and establish an opening through the skin and subcutaneous tissue.

DISCUSSION

Dr. George Fetterolf: What makes that baby breathe the way he does?

Dr. Oram R. Kline: There is partial atresia of the posterior nares. We have never been able to get a probe through the left side, and with difficulty through the right side. I do not believe the child breathes through the nose at all, although some air does get through the nose. The noisy breathing is due to oral respirations in such a young child. There may be an enlarged thymus there.

Dr. Fetterolf: Have you examined the root of the tongue to see if there is a lingual thyroid there?

Dr. Kline: No.

Granuloma Inguinale, with Associated Lesions of the Lip, Pharynx and Larynx: Report of a Case

BY R. J. HUNTER, M.D.

Granuloma inguinale is a disease endemic in this latitude which has only recently been recognized in Philadelphia, as shown in an article by Randall, Small and Belk, published in June, 1922.[1] These cases have been treated for many years at the Philadelphia General Hospital with little or no improvement, diagnosed as tuberculosis cutis, obstinate chancrodial or syphilitic infection. It is interesting to note, however, that Dr. Stelwagon,[2] on duty many years in the skin wards of this hospital, remarked upon the similarity as follows, speaking of granuloma tropicum: "It has some features of a cutaneous tuberculosis with an added pus cocci infection. In some respects the creeping and undermining ulcerating tract, starting from a gonorrheal or chancroidal bubo in the groin, in a tuberculosis patient, occasionally seen (formerly more than now) in our Charity Hospital venereal wards, considerably resembles it." All of the cases seen at this hospital have been in the negro race.

The lesion begins as a firm, dome-shaped papule over which the superficial layers of the skin appear smooth and atrophic. Later, from pressure atrophy, a small ulceration in which granulation tissue promptly appears, occurs over the center of the papule. The lesion gradually extends until large areas of granulations may develop. Accompanying the lesions, especially those of the prepuce, scrotum or labia majora, there may develop a marked brawny edema, at times suggesting elephantiasis. It is most frequently found in the inguinal region, but other adjacent parts have been involved. There is little tendency to heal, although the older lesions show some effort at cicatrization in one direction with extension in the other. The process goes on almost indefinitely,

[1] Surg., Gynec. and Obst., June, 1922, 717–739.
[2] Diseases of the Skin, 1910 edition.

and until the true diagnosis was established the cases showed little or no improvement with any treatment.

The diagnosis is established by finding the specific organism as originally described by Donovan and by the immediate and spectacular effect of the specific remedy, antimony, usually employed in the form of tartar emetic, given intravenously in doses beginning at 5 cc of a 1 per cent solution and increasing 1 cc daily until 10 cc are reached, then 10 cc every other day. For microscopical diagnosis, direct smear preparations of the exudate from the surface of the lesions are most satisfactory. These are stained rather intensely with Wright stain. Two features of such smears are characteristic: (*a*) The appearance of considerable numbers of large mononuclear cells, and (*b*) the presence of encapsulated bacillary organisms within the cytoplasm of these mononuclear cells. These organisms are thought to be bacteria of the Bacillus mucosus capsulatis group. It is noteworthy that in rhinoscleroma the typical intracellular organism, Bacillus rhinoscleromatis, belongs to the same group of bacteria.

The case we have to report is not included among those in the previous article. One point of interest to this Section is due to the fact that it is the first case we know of in which lesions of the pharynx and larynx have been associated with the inguinal condition. Three previous cases have been reported with lesions of the lip.[3, 4] Our case was admitted to the hospital September 9, 1920, with ulcerating lesions of the groin, lip and pharynx. His Wassermann was negative with Noguchi and luetic antigens and plus 4 with cholesterin antigen. The lesions were considered syphilitic and he was given four injections of 0.9 gm. of neoarsphenamine, without apparent result. Four months previously he had received five injections of arsphenamine. He left the hospital on a pass and did not return until November 2, 1922, when the diagnosis of granuloma inguinale was made, by demonstrating the organisms in smears from lesions of the groin, perineum

[3] Beeson, B. B.: Arch. Dermat. and Syph., 1922, vi, 342. Tarra's case, cited by Beeson.

[4] Sequeira: British Med. Jour., March 17, 1908.

and lip. As he was unable to open his mouth we could not see the lesions in the pharynx and larynx. His Wassermann was negative, but in spite of this fact, to completely eliminate the possibility of syphilis, he was given three doses of 0.9 gm. of neoarsphenamine and 1 teaspoonful of mixed treatment, three times a day, with absolutely no effect. He was then put on intravenous injections of tartar emetic and the lesions healed, improvement being noticed after the third injection. His voice, which had been extremely hoarse, improved, and he was able to take food much better. Although we were unable to reach the lesions in the pharynx and larynx to make smears, we feel that the fact that the organism was found in the lip lesion and in the groin, together with the fact that the dysphagia, laryngeal and external lesions showed no improvement under antisyphilitic treatments, but cleared up promptly with tartar emetic, is *prima facie* evidence that the lesions in the pharynx and larynx were also due to the organism of granuloma inguinale.

This patient has cicatricial stenosis of the velum of the palate. The uvula has disappeared and the tonsils seem completely atrophied or removed. No pressure was made on the pillars to determine the depth of the fossæ or presence of tonsillar remnants. The palate is adherent to the posterior pharyngeal wall, and there is an opening, 3 cm. long by 2 cm. wide, leading to the epipharynx. The epiglottis has either disappeared or is included in a cicatrix which leaves the introitus as an oval opening about 4 x 3 cm. in diameter. No view of the cords could be obtained. Instead of the ordinary view of the larynx, one simply looks down through this circular opening. This patient is in condition for operation, and it is presumed that he will be returned for plastic operation on the palate and treatment for stenosis of the larynx. There was some bilateral ankylosis of the jaw.

Case History (on First Admission);

G. B., negro, aged twenty-nine years. Admitted to Philadelphia General Hospital, September, 9, 1920.

Chief Complaint. Sore throat. Sore genitals.

Family History. Negative.

Past History. Ordinary diseases of childhood. Has always been healthy. Had a sore on the penis four years ago which healed under treatment.

Personal Inspection. Noticed that voice began to fail four months ago. He received five intravenous injections of "606" at that time. He has had a sore in the inguinal region for about two years.

Physical Examination. Fairly well-built black male, about thirty years old. Head and neck. Upper lip shows a large, hard, saucer-shaped, sloughing ulcer.

Mouth: Talks in a whisper. Throat shows uvula sloughed away and ulceration on the posterior pharyngeal wall. Chest negative. Abdomen negative.

Genitalia: Beginning in inguinal region and extending downward under the scrotum is a reddened, ulcerating and constructive growth, which shows peripheral healing and central sloughing. Where the tissue has not sloughed is a grayish, dry, elevated growth which resembles dead epithelium.

Penis: Phimosis present and a hard, indurated foreskin.

Diagnosis (I): Tuberculosis cutis.

Alternate Diagnosis: Tertiary syphilis. (Signed, Andrew.)

Laboratory Findings: Urine straw colored; no sediment; acid reaction. Specific gravity, 1020. Sugar, negative. Albumin, negative. Microscopical examination, negative. September 20, 1921, blood Wassermann, luetic and Noguchi antigens, negative. Cholesterin antigen: Plus 4.

Treatment, September 20 to 27: Potassium iodide, gr. x, three times a day. Dobell's (gargle), September 27 to November 29, mixed treatment, 1 teaspoonful, three times a day. September 20, neoarsphenamine, 0.9 gm. September 28, neoarsphenamine, 9 gm. October 5, 0.9 gm. neoarsphenamine. October 12, neoarsphenamine, 0.9 gm.

Patient went out on pass and never returned. Throat condition much better.

CASE HISTORY (on Second Admission):

G. B., negro, aged thirty-one years, laborer, admitted November 2, 1922.

Report from receiving ward, November 2, 1922.

Patient gives history of primary sore about six years ago. He has been treated, including several injections in the arm. He received his last injection about one year ago. About three years ago large buboes developed, and they have been operated upon several times, with incisions extending back to the anus. His scrotum has been swollen for several months. He has had the ulcerated condition of the upper lip for about two years and has had extreme hoarseness and sores in the mouth for two or three months.

Impression: Lues and luetic granuloma.

Primary History of Condition upon Admission. Ward, venereal. G. B., negro, aged thirty-one years. Birthplace, South Carolina. Occupation, laborer. Admitted, November 2, 1922. Attending physician, Dr. Kirby. Intern, Dr. Blackmon.

Chief Complaint. Sore on upper lip, right and left sides of scrotum externally; hoarseness.

History of Present Illness. About three or four years ago patient was operated on for buboes. After this the incision healed up in about a month, but shortly afterward ulcerative areas began to appear, but more marked on the right side. These spread quite rapidly and went almost to the rectum. He says he was operated on several times for this condition, and it was apparently healed after several months of treatment and remained so for over a year. He came to this hospital about two years ago and the condition was practically healed, when he had to go home, where he stayed. For the last five months the ulceration has been spreading very slowly.

The ulceration on the upper lip began a little over a year ago as three small sores. These became larger, and in a few months all of the upper lip was involved. He received treatment for some time and the condition has remained the same for the last few months.

The hoarseness appeared about the same time as the ulceration on the upper lip. At first he could talk all right at times, but finally he became hoarse and has been so since then. He says that his throat was sore at first, but this gradually disappeared and is practically painless at present, except on coughing.

Family History. Mother died of "stroke;" father dead, cause unknown. Two brothers living and well; two brothers dead, causes unknown; no history of tuberculosis, cardiorenal disease or malignancy.

Personal History. Measles, whooping-cough, mumps during childhood. Pneumonia at age of seventeen. Chancre about six years ago; gonorrhea about three years ago.

Physical Examination. Fairly well-developed adult male negro who shows evidence of loss of weight. Extensive area of ulceration involving the lower portion of the upper lip.

Head. Scalp: Negative.

Eyes: Right pupil reacts fair to light; left reacts less and has small opaque area present. No nystagmus or strabismus, but slight ptosis of both upper lids.

Nose: Hypertrophied turbinate anteriorly on the left.

Ear: Negative from external examination.

Mouth: Ulcerative area with well-defined edges involving the lower part of the upper lip. No discharge and partly covered with granulation tissue. Teeth in fair condition; tongue slightly coated. Further examination unsatisfactory, as patient is unable to open mouth very well.

Neck: Posterior cervical glands enlarged. Visible throughout. Otherwise negative.

Chest. Lungs: Breath sounds somewhat harsh throughout. Otherwise negative.

Heart: Systolic roughening in mitral area. Otherwise negative.

Abdomen: Liver enlarged to one finger's breadth below the costal margin. Otherwise negative.

Scar in both inguinal regions, more marked on the right. Area of ulceration on each side just lateral to the scrotum and on the

perineum from scrotum to anus. Area is moist and somewhat covered with granulation tissue. Irregular in outline.

Extremities: Negative except for showing emaciation.

Impression: Granuloma inguinale; lues; syphilitic laryngitis (?). (Blackmon.)

December 30, 1922. The lesions on the upper lip and in the perineal region were both positive for granuloma organisms. That on the lip did not resemble granuloma in appearance, however. To determine if antisyphilitic treatment would help

SOLUTION OF TARTAR EMETIC 1 PER CENT GIVEN INTRAVENOUSLY.

Date.	Amount.
November 28, 1922	5 cc
November 29, 1922	6 cc
December 1, 1922	7 cc
December 2, 1922	8 cc
December 4, 1922	9 cc
December 5, 1922	10 cc
December 6, 1922	10 cc
December 8, 1922	10 cc
December 11, 1922	10 cc
December 13, 1922	10 cc
December 15, 1922	10 cc
December 17, 1922	10 cc
December 19, 1922	10 cc
December 21, 1922	10 cc
December 23, 1922	10 cc
December 26, 1922	10 cc
December 28, 1922	None[1]
December 30, 1922	10 cc

[1] Pains in shoulders, weakness and dizziness.

November 7, 1922. Request for consultation with laryngologist:

With reference to examination of larynx: Patient has had chronic hoarseness for two years. History of lues. At present has granuloma inguinale. (Drs. Kirby and Blackmon.)

November 9, 1922. Impossible to make laryngeal examination, due to the fact that the patient cannot open his mouth sufficiently. (Dr. Keenan.)

February 5, 1923. Nasal septum deviated, obstructing the left side; ear-drums thickened, not retracted. Hearing normal. This patient has cicatricial stenosis of the velum of the palate. The uvula has disappeared and the tonsils seem completely atrophied or removed. No pressure was made on pillars to determine depth of fossæ for the presence of tonsillar remnants. The palate is adherent to posterior pharyngeal wall and there is an opening, 3 cm. long by 2 cm. wide, leading to the epipharynx. The epiglottis has either disappeared or is included in a cicatrix which leaves the introitus as an oval opening about 4 × 3 cm. in diameter. No view of cords could be obtained. Instead of the ordinary view of the larynx, one simply looks down through this circular opening. Advise when patient is in condition for operation that he be returned for plastic operation on the palate and treatment for stenosis of the larynx. Some bilateral ankylosis of the jaw. (Dr. Hunter.)

his voice or the lesion on the lip, this was given over a period of three weeks, but it affected neither. Tartar emetic was given intravenously as recorded below, and after the third injection improvement was noted. That on the lip improved markedly until after the seventh injection, then no change was noted until December 15, 1922, when it was kept moist with glycerin and healing took place rapidly. The cause for the chronic hoarseness has not as yet been determined, although there are no physical signs, as far as I can find, of an aneurysm. It would be a good idea to have his chest *x*-rayed to see if there be anything causing pressure on recurrent laryngeal nerve. No examination can be made of the larynx due to inability of the patient to open his mouth. (Blackmon.)

Treatment, November 11, 1922. Mixed treatment, 1 teaspoonful three times a day. Discontinued November 17, 1922.

November 17, 24 and 29. Neoarsphenamine, 0.9 gm. (three doses).

February 27, 1923. Patient much improved. All former areas of ulceration now healed by scar formation.

Final Diagnosis: Granuloma inguinale.

DISCUSSION

Dr. B. Alexander Randall: I would like to point out six or seven interesting features in regard to this topic.

First of all, the finding of so many cases of granuloma in Philadelphia, as published in our recent series, is rather startling. Our first thought was to verify the accuracy of these diagnoses, and we went back to the original description as given by Vianna in his studies at the Institute of Oswaldo Cruz, in Rio Janeiro, and were able to completely substantiate the diagnostic features as originally described by him.

Second, the finding of granuloma here in Philadelphia immediately took the disease out of the category of "tropical diseases" in which, for a long time, it has been classified. Out of our 16 cases, 14 were residents of the vicinity of Philadelphia, and only 2 had at any time been south of Mason and Dixon's Line, so we must consider it a newly recognized disease in our midst.

The third thing of particular interest is, that if these cases are present in Philadelphia now they must have been in the past, and looking back over the records of Blockley we found numerous similar cases masquerading under different diagnoses as far back as we care to go. The records of Dr. Horwitz, twenty-five years ago, with photographs of cases have been found, each representing the lesion as we know it today.

The fourth interesting feature is the various diagnoses under which it has been treated. Dr. Horwitz called the condition "Tuberculosis Cutis," and treatment instituted by him has been in vogue ever since. It consists principally of heliotherapy. Dr. Rosenberger and the late Dr. Uhle, in 1912, attempted to prove the tuberculous character of this lesion, but failing to establish this diagnosis, went no further with the work. A great many cases have been treated as being luetic; others as phagedenic chancroids, and still others as condylomas.

The fifth interesting thing is that in our experience the disease has been practically limited to the negro. I do not believe we have seen a single clear-cut definite case in a white man, though 1 such case has been reported in England which was contracted in Brazil.

The sixth interesting point is concerning the bacteriological work. Dr. Small has carried on a most painstaking and far-reaching research as regards the etiological organisms, and I will leave to him the discussion of this phase.

The seventh interesting thing is in regard to the treatment. Even if the condition had been recognized prior to 1910, that is before the time when intravenous therapy was developed, I doubt if its treatment would have given the startling results which we have seen following the intravenous administration of tartar emetic. I have seen more brilliant healing of these lesions under this treatment than we have seen following the use of salvarsan in the long-standing, syphilitic skin lesion. We have used the drug in a 1 per cent solution with a maximum dose of 10 cc. We know that we are dealing with a toxic drug, but as yet have not seen any serious results. At times cardiac embarrassment, respiratory difficulty and deep-seated bone pains have been encountered.

The eighth and last interesting feature is a recent one, where we have been using two new antimony compounds in the hope to get away from the toxic symptoms at times following the administration of tartar emetic. Through the courtesy of Dr. Abel, of Johns Hopkins Medical School, two new antimony synthetics have been placed at our disposal, and they have proved to be of high therapeutic value and low toxic reaction when used intravenously; and of still greater importance is the fact that just within the past two weeks we have found that we can use these new drugs as an intramuscular injection, having attempted the same in a case where

intravenous therapy became impossible—in fact, we have now tried it intramuscularly in 3 cases without apparent local reaction.

Dr. James C. Small: I have not very much to add. My viewpoint has been that of the laboratory man and concerns the question of diagnosis. I have seen in the neighborhood of 40 cases of this disease. Cultures have not been made from all of them. The organisms we find here are bacteria. They are, I believe, bacilli of the same general type to which the Friedländer and rhinoscleroma organisms would belong. The unfortunate part of the whole thing is that we have not been able to reproduce these lesions experimentally, and therefore a doubt remains as to whether the organisms grown in cultures are the causative agents of this disease. Further work along this line will be necessary, and, of course, it is not at all unusual to be unable to produce a lesion in an animal which will resemble a lesion in man. A thing of special interest to mention and emphasize is that the organisms found in rhinoscleroma are of the same general type, and that they occur within mononuclear cells.

At a meeting in New York last year, it was suggested that tartar emetic might be of therapeutic advantage in rhinoscleroma.

There is another condition that I have thought of in this connection, and that is the condition known as trachoma. In this condition we have as a diagnostic finding the intracellular bodies. They differ somewhat from the organisms we find here, but they might be of the same general type. I do not know whether antimony has ever been tried in trachoma or not. It might be worthy of a trial.

If we have in tartar emetic an agent which is effective against a bacterial infection, as far as I know, it is the first chemical therapeutic agent that can be used and applied in a practical way in dealing with a bacterial infection.

Dr. J. A. Kolmer: I am very much interested in hearing Dr. Small's opinion of this organism, and if it is a bacterium rather than a protozoön, as he has stated, it is probably the most striking instance of successful chemotherapy which we have in the treatment of a bacterial infection. The only other drug that commands any attention in this respect is ethylhydrocuprein for pneumococcus infections.

I would suggest that it might be worth while to use tartar emetic in the treatment of ozena, because the bacillus of Perez, which is regarded by some as the cause of ozena, belongs to the same mucosus group as the bacillus of rhinoscleroma, and presumably of this bacillus of granuloma inguinale.

I would like to ask Dr. Randall the name of the second preparation that has been used by Dr. Abel for intramuscular injections without irritation.

Dr. Randall: Both drugs have been used successfully, both intravenously and intramuscularly.

Tuberculous Lesions of the Ear, Nose and Throat

BY CURTIS C. EVES, M.D.

This patient, F. A. J., male, aged thirty-one years, presents lesions following tuberculosis of the lungs, the ears, the larynx and the nose. After the disease has nearly destroyed the function of the ears and larynx, it has become quiescent and is now active in the nose only. In none of these conditions has tubercular bacillus been demonstrated. The clinical picture shown has been clear enough in each case to warrant a belief that it is tubercular in origin. A Wassermann test of his blood proved negative.

Tuberculosis of the lungs was first positively diagnosed in 1909 while he was a junior in the engineering course at State College. After a two years' rest-cure at home and in New Mexico, he was well enough to finish his course at the School of Mines in New Mexico. The left ear began suppurating, without other symptoms, in 1911. The discharge continued for several months with a gradual diminution of hearing and almost entire destruction of the drum membrane. In 1918, the same condition appeared in the right ear. Both ears, after discharging, nearly dried up, leaving the patient so deafened that he can only hear by aid of artificial means. Three or four weeks after the first ear began, in 1911, the patient began to lose his voice. There was no pain or discomfort at the time.

I first saw the patient in the spring of 1914, when he consulted me for treatment of the larynx. At that time the left arytenoid was swollen, with considerable thickening in the interarytenoid space. The margins of the cords were irregular and thickened. The following year he returned to New Mexico, where, after making

a considerable study of the conditions, he treated his own larynx by means of reflected sun rays (heliotherapy), which after a long period of patient practise he claims to have cured the thickening on the arytenoid. This, however, did not restore his speaking voice. (The patient, being well educated, and the son of a doctor, has made a very careful study of his case. He has published a very intelligent article upon heliotherapy of the larynx.)

The present condition of the nose began in 1920, when I first noticed a small indurated ulcer upon the right side of the septum, the patient having then consulted me for a nasal obstruction which he claims dated back previous to 1911, when a doctor in New Mexico had taken out a greater part of his middle turbinate. At the time of his visit and for three or four subsequent visits I treated the ulcer with solution of silver nitrate. Six months later, the ulcer having increased in size, it was treated by several cautery punctures made into the surrounding thickened mucous membranes. At three different times the cautery puncture was used at from two to three weeks' intervals.

After a period of several months the patient again returned; the ulcerations showed increasing area and a new ulcer was developing higher up on the same side of the septum. On January 17 and 24, 1923, 25 mg. of radium were applied. The patient thinks the condition has improved since these treatments.

There has been no apparent difference in the condition; an ulcer developed on the other side of the septum since the treatment of January 17, 1923. During the period between the first few treatments with silver nitrate in 1920 and the thermocautery in 1921, the patient applied the sun's rays to the nasal ulcer to the extent of causing soreness and an increase in the size of the area, without beneficial effect.

I only saw the patient once during the active state of the suppuration of the right ear, at which time the membrana tympani was almost entirely destroyed and only a slight mucopurulent discharge was present, which showed no tubercular bacillus under microscopical examination.

Report of a Series of Cases of Asthma, with Special Reference to Diagnosis and Treatment

BY HARRY B. WILMER, M.D.

Before presenting to you the contents of this paper, may I state that I am an internist and am not versed in the finer arts of rhinology, otology and laryngology. May I, therefore, ask your forbearance if I sound somewhat primitive at times in my expression.

The purpose of this paper and the presentation of these case reports is to try, if possible, to demonstrate to you how valuable the cutaneous test may be in diagnosing the cause of asthma and in treating the disease.

I feel sure that many cases operated upon for deflected septum, polyps, enlarged ethmoids, etc., could have been spared these operations and have obtained better results had a thorough cutaneous test been performed. It would be a presumption for me to suggest in what types of cases operation is indicated, but I do feel that no one suffering with bronchial asthma should be operated upon until an exhaustive history has been obtained and a thorough cutaneous test performed.

In order to support the above statement, let me cite the following statistics: (Quoting from L. C. Walker's work in Volume 3, of the Oxford Loose-leaf Medicine).

In 150 cases studied by Walker, of the Peter Bent Brigham Hospital, 55 per cent were found sensitive to some protein. Of this group horse dander was the cause in about 20 per cent, wheat protein 15 per cent, bacteria 15 per cent, pollens of spring flowers 15 per cent, pollens of autumnal flowers 10 per cent, and cat hair 5 per cent. In my own experience, after reviewing a series of 100 cases, I have found that 70 per cent were found sensitive to some protein. Of this group, chicken feathers and goose feathers were the cause in about 20 per cent, eggs and milk 15 per cent, wheat 10 per cent, spring pollen 5 per cent, fall pollen 10 per

cent, horse dander 2 per cent, house dust 3 per cent, tomatoes 2 per cent, dog hair 1 per cent, cat hair 1 per cent, and rabbit hair 1 per cent.

In the *American Journal of Diseases of Children*, September, 1922, Harold Herman reports a series of 31 children suffering with bronchial asthma. Of these 31, 11 showed absolutely no positive reactions and 3 others only such a slight reaction as to be considered, finally, non-sensitive. The remaining 17 gave 82 positive reactions and 170 positive and doubtful together. This makes an average of 5 positives per patient and 10 positive or doubtful. There were 4 with only 1 positive, 5 with only 2 positives and 1 with only 3 positives, the remainder all having more than 3 and in 1 case as many as 17 positive reactions.

Brodie and Dixon have shown that the vagus is the only motor nerve to the bronchial muscles, and that in this nerve run two sets of fibers, the constrictor and the dilator.

Examination of the sympathetic gave negative results so far as the bronchial muscles were concerned. They found that stimulation of the constrictor fibers of the vagus could be excited by various irritants of the nerve itself or of the respiratory mucous membrane, and a typical attack of asthma would result: prolonged expiration, distended lungs, dry rales, etc.

Sewall has shown that guinea-pigs, especially after previous sensitization, may react to intranasal instillations of horse serum by the development of a typical attack of bronchial asthma.

Therefore, by putting these findings together, it is shown that protein applied to the upper respiratory tract of a sensitive animal (Sewall) irritates the constrictor fibers of the vagus (Brodie and Dixon) producing stenosis of the small bronchi by causing a spasm of their circular muscles (Auer and Lewis). The mechanism of a typical attack of true asthma is thus explained.

The diagnosis of the cause of bronchial asthma is not an easy task in the majority of cases, and not infrequently all the resources of the diagnostician are taxed to the limit; but if the following rule is adhered to religiously, a diagnosis can be made in from 55 to 70 per cent of cases:

(*a*) A thorough and exhaustive history. Much can be learned from the history as to the patient's habits, his environment, his occupation, his food and whether his attacks are seasonal or perennial in type.

(*b*) A thorough questioning as to the previous medical history. I have found a large percentage of cases giving a history of influenza, severe bronchitis and, in a few cases pregnancy, preceding the first attack of asthma.

(*c*) A complete *x*-ray study of the sinuses, especially if a headache is a constant symptom.

(*d*) If there are symptoms of nasal obstruction, or if there is constant sneezing accompanying the attack, a thorough nasal and laryngeal examination should be requested.

(*e*) A thorough cutaneous test with all the proteins and pollens should be performed. The bacterial proteins I have practically discontinued using, due to the fact that I have found them, in many cases, unreliable and misleading.

If a case does not react to any protein or pollen it has been my practice to classify that case as a bacterial case until proven otherwise. Here let me emphasize the importance of a thorough cutaneous test. I have on several occasions had patients tell me that they had been tested before coming to me, and on questioning these patients I have found that they had had several proteins applied and had been told that there was no evidence of protein sensitivity. In other words, these patients had not been thoroughly tested, due to the fact that the person doing it was not equipped with sufficient material to carry on the test thoroughly. No patient should be considered non-sensitive until every available protein or pollen has been tried.

The cutaneous test is just as important in helping to diagnose the cause of a bronchial asthma as any other method of precision used in diagnosing another form of disease, and when half-way done is more dangerous to the patient than if it had never been done at all.

For the sake of convenience in diagnosing, let me divide the causes of bronchial asthma into the following groups:

1. *The Thermal Type.* In this type the patient complains of the asthmatic attacks occurring immediately upon leaving a warm or hot room and going into the cold, or *vice versa;* and, again, it may be only a warm or cold draft which will precipitate an attack; or he may be walking in a cold wind, and thus bring on an attack. In this type of cases it has been my experience that the patient has been at some time in the past, or is at the present, sensitive to some pollen or animal emanations and has developed a definite bacterial sensitivity which has become engrafted upon an already sensitive mucous membrane. I have never been able to find, through the cutaneous test, one of these cases sensitive to any food protein.

Miss L. M., aged forty-one years. Chief complaint, asthma, constant sneezing and marked coryza.

Present Illness. Twenty-five years ago patient had an attack of rose cold which has persisted until six years ago, when the hay fever symptoms disappeared. Since that time the only symptoms are constant sneezing, coryza and asthmatic attacks brought on by sudden changes in temperature and especially on leaving the warm office and going out into the cold air outside. Also attacks may be brought on by sitting in the open trolley.

Social History. Mother has had hay fever, but no other members of the family have been afflicted.

She has had several operations upon the nose at different times, but has found no relief.

On performing the cutaneous test upon this patient, I found that timothy reacted only slightly, but no other reactions showed, as this patient presented herself for treatment in the late fall of 1921. I decided to try an autogenous bacterin made from the nasal mucous membrane, which was given with complete relief of symptoms in six weeks from time of starting treatment.

2. *The Chemical Type.* This is the type of patient who develops an attack of asthma on inhaling any pungent odor, such as highly perfumed soaps, cologne, tobacco smoke or highly scented flowers, or if any chemical is applied to the mucous membrane. In this

type of cases, as in the thermal type, I have never seen a case that did not show a sensitivity or give a history of the same to the pollens, animal emanations or house dirt.

Mrs. N., aged thirty-six years. Chief complaint, asthma of ten months' standing.

Present Illness. Started ten months ago during an attack of grippe and severe bronchitis.

The attacks are intermittent, occurring only when she feels chilly or as if an attack of grippe were coming on.

She has been in the habit of taking a grippe capsule which had been prescribed for her and immediately the attack of asthma would appear.

Rx aspirin was found to be contained therein.

Upon instruction to never take aspirin again, the patient has been free of asthma.

3. *The Neurotic and Atmospheric Type.* In this type the patient is highly neurotic and asthmatic attacks are precipitated by fright, sorrow, hysterics or any other sudden emotional outbreak; and in the atmospheric type is seen the patient who develops an attack just before a thunder storm or on any sudden change of atmospheric pressure. These two types are extremely difficult to diagnose, but my experience has been that these patients, in the majority of cases, have had or have a sensitivity to the animal proteins and pollens plus a markedly depleted physical condition, due to some long-standing infection or great mental strain.

4. *The Pollen Type.* There is little to say concerning this type, as the cause is easily found by the cutaneous test. This type, however, may be perennial as well as seasonal. Do not overlook the fact that there is, in almost all of these cases, a secondary bacterial infection of the mucous membrane, and I believe that in almost every instance the cause of the asthmatic attacks in the pollen type is due to bacterial invasion plus the presence of the pollen itself—I feel that the constant bombardment of the mucous membrane by the pollen lowers the physical resistance of the patient and the mucous membrane is made a fair field on

which the bacteria may grow, and, therefore, the asthmatic attacks appear. In order to bear out my theory, I should add that asthmatic attacks are rarely seen occurring until at least three or four weeks have elapsed after the hay fever begins.

5. *The Animal, Epidermal and Food Protein Type.* In this type are classified the patients sensitive to the food and animal proteins. If the asthmatic attack is caused by the animal protein it will readily be shown by the cutaneous test. On the other hand, if it be food protein sensitivity the offending protein is not always easy to locate; and as I have said before, an exhaustive study should be made before the patient is considered non-sensitive. It must be borne in mind that absolute withdrawal of food from the diet may give a negative test. This should be remembered, as the evidence that a particular food is causing the trouble may be so great that that food will be withdrawn and then a later test may give a negative reaction to that food.

6. *The Bacterial Type.* In this type of patient the cutaneous test is negative throughout, although there may be a history of hay fever attacks or asthmatic attacks being caused by contact with animals. Almost invariably there is a history of influenza or some acute infectious disease, preceding the first attack of asthma. The method of diagnosis in these cases is one of elimination alone, and no case should be diagnosed a bacterial case until all methods of diagnosis have been exhausted. There is no doubt in my mind, however, that a bacterial sensitivity may be engrafted upon or may be the direct cause of asthma in any of the foregoing types classified above.

Treatment. Let us now consider the treatment of these different types of bronchial asthma. The treatment of the thermal and chemical type is practically the same. Therefore, let us consider them together. If the cutaneous test shows a positive reaction to any of the animal proteins it is my habit to first give 10 gr. of potassium iodide, three times a day, for three or four days, as the drug thins the secretion and allows free drainage from the bronchi, thereby increasing the chances of recovery of the offending bacterial organism. An autogenous bacterin is then made and immediately the offending animal protein is injected

in ascending doses. If after a period of three or four weeks there is no sign of relief the bacterin is given in conjunction with the protein extract every five days. The accompanying charts will show the percentage of cures, etc. If the cutaneous test is negative a course of bacterin injections is given with a general supportive treatment.

In 5 cases of asthma of the thermal and chemical types who had been tested cutaneously there was a definite pollen sensitivity without symptoms of hay fever. These cases were treated with the respective pollens causing the reactions, and gave, in 3 cases, a 100 per cent relief, in 1 case a 75 per cent relief and in 1 case no relief.

In the pollen type of asthma the treatment is comparatively easy, as in the majority of cases the hay fever symptoms are also present and the treatment is the full course of injections of the offending pollen. In the average case these injections will take care of the asthmatic attacks. If, however, the history shows that in previous years the asthmatic attacks have been extremely severe and have continued long after the hay fever symptoms have subsided or disappeared, or if the only remaining symptom is a cough or constant sneezing, an autogenous vaccine is made from the sputum or from the nasal mucous membrane and the bacterin is used in conjunction with the pollen extract injections, with most satisfactory results.

Last year I saw 1 case in which the only symptoms were those of asthma. This asthma occurred and had occurred for several years, beginning about August 20, and lasting through the fall hay fever season. On testing this patient for the fall hay fever pollens I was surprised to find that she did not react to anything. I therefore took for granted that the patient had sufficient resistance developed to show no sensitiveness to the cutaneous test, so I gave her a full course of ragweed injections, with the result that the patient went through the season without one attack of asthma.

Often these pollen cases will be left with a definite sensitiveness of the nasal mucous membrane after the season. In these cases it has been my practice to have an autogenous bacterin made

from the nasal secretions, and eight to ten of the injections of bacterin have given in the majority of cases the desired results.

Let us now consider the treatment of the protein type of asthma. In this type we will take up both the food proteins and animal proteins causing asthma.

Where the asthma has been the result of some food protein, as is determined by the cutaneous test, the food should be immediately removed from that patient's diet. In a few cases in adults there will be an immediate relief of symptoms, but in the majority of cases no relief will be experienced, especially in the aged and in the cases that have been long standing. The reason for this is, I believe, that there is a secondary bacterial infection engrafted upon the already sensitive mucous membrane of the nasal and laryngeal passages. Here again the bacterin is brought into play as a curative agent.

In children when there is a definite sensitivity to food which has not been of long standing the removal of that food will in many cases completely cure the condition of asthma.

I have never been able in my experience to get any patient to undertake the desensitization treatment against a food sensitivity, due to the long course of treatment which is necessary.

Considering the animal protein type of asthma, it is usually customary to remove the offending protein, such as chicken feathers, goose feathers, cat, dog, etc.; but where the animal protein cannot be removed, due to a patient's employment, it is then necessary to desensitize that patient by a full course of that animal protein. In 9 cases of horse asthma treated in this manner I have had 100 per cent cure in all 9 cases.

At this point I would like to report a case that to me was most interesting, and will show the multiplicity of reactions:

A. B., aged thirteen years, gave the following history: Hay fever at the age of five, asthma at seven and eczema at eight. On testing this boy I used all of the pollens and found that he reacted to ragweed, short and long, and all the fall pollens, also very lightly to three of the spring pollens, I then tested him with the

animal proteins and found that he reacted to chicken and goose feathers and slightly to dog and cat. He did not react to any of the food proteins. There was a dog next door with which the

A SERIES OF 35 CASES OF ASTHMA TREATED WITH AUTOGENOUS BACTERINS ONLY.

Relieved.			Marked improvement.			Not benefited.		
No. of patients.	Age of patients.	Duration of disease.	No. of patients.	Age of patients.	Duration of disease.	No. of patients.	Age of patients.	Duration of disease.
1	4	2	1	26	4	1	28	2
1	5	2	1	28	10	1	52	10
1	7	6½	1	35	15	1	52	20
1	7	4	1	35	8	1	58	30
1	7	4	1	40	15	1	58	30
1	8	6	1	40	20	1	58	20
1	20	10	1	43	7	1	59	20
1	28	3	1	43	3	1	48	15
1	30	4	1	60	20	1	49	30
1	32	10						
1	40	20						
1	42	2						
1	46	5						
1	50	1						
1	53	20						
1	60	25						
1	63	18						

SERIES OF 100 CASES OF ASTHMA TREATED WITH PROTEINS AND BACTERINS COMBINED.

Age.		1–10	10–20	20–30	30–40	40–50	50–60	60–70	
No. of cases		**10**	**10**	**20**	**25**	**20**	**10**	**5**	**100**
Chemical		..	..	..	3	1	..	..	4
Thermal		..	..	2	2	2	1	..	7
Pollen		3	5	10	11	5	4	1	39
Animal		2	3	6	6	3	2	1	23
Food		6	5	6	6	5	2	..	30
Bacterial		3	2	6	7	9	6	4	37
Result of Treatment.	Pollen	3	5	10	11	5	4	1	39
	Bacterial	5	5	6	12	14	8	5	55
	Animal	2	1	3	1	1	..	..	8
	No result	..	..	**2**	**4**	**4**	**3**	**2**	**15**
	50%	..	..	**4**	**2**	**1**	**2**	..	**9**
	75%	**1**	**1**	**3**	**5**	**1**	**2**	**2**	**15**
	100%	**9**	**9**	**11**	**15**	**13**	**3**	**1**	**61**

boy played occasionally, but no cat. We removed the pillows and quilt which were upon the bed and last year desensitized him with ragweed, both long and short, with the result that the child's eczema entirely disappeared, also his asthma, and he had a 100 per cent hay fever cure.

In any of the animal protein types of asthma I have not had to use a bacterin in addition to the animal proteins used in treatment. These types of asthma have been the most spectacular in their sudden cures. Here again the cutaneous test is invaluable.

In the bacterial type of asthma the treatment is not so encouraging, as the results are variable. In a series of 35 cases of asthma of the bacterial type treated last year I had 17 cases completely cured, 9 markedly benefited, and 9 not benefited. The percentage is about the same as that of Dr. I. Chandler Walker's, of Boston, the bacterin treatment in these cases being as follows:

The patient is given immediately a saturated solution of potassium iodide and is instructed to take 15 drops, three times a day for at least a week before a sputum is collected. The sputum is then collected and cultured and all organisms showing in the culture are used in the making of the bacterin. The bacterin is then made up in a dosage of minims and each minim contains approximately 1 to 2 million organisms. The injections are given every four days, increasing 1 minim a dose. In a small percentage of cases of this type, especially in the aged, and in the long-standing cases, there will be a complete remission of symptoms for periods ranging from three months to a year. These people have never previously been free from attacks. They will then return for a series of four or five injections in larger doses, and most of the cases who have thus returned have not, to date, been seen for further treatment.

DISCUSSION

Dr. J. A. Kolmer: It is very difficult, indeed, to add anything to Dr. Wilmer's very excellent presentation this evening. I by no means have had the experience upon which to base my discussion that he has

had. I think, however, there is one outstanding feature, and that is the fact that all cases of asthma that consult the physician are worthy of investigation from the standpoint of allergin sensitization.

Possibly only those cases due to cardiac disease can safely be omitted from a study of this sort. I know of no human disease in which the results have been more gratifying than in the treatment of allergic asthma.

I think the second fact that stands out is that the majority of us are not in a position to give these cases the study that they may demand. We have to look upon one or two physicians in a community who will equip themselves with the large list of protein allergins that are necessary to conduct these tests. That is one of the discouraging features. Every year our list of allergins is added to. We wonder when we are ever going to reach the end of the allergins that we must keep on hand to give these individuals a thorough study. As far as asthma has been concerned, it has been largely confined to children. You know the late Dr. Meltzer, of the Rockefeller Institute, is to be given the credit for showing us the allergic nature of asthma, and he noted that in the guinea-pig this animal died practically with an asthmatic attack, with an enormous emphysema of both lungs, and it is true that the human being tends to act in this same way. The poison, whatever it is, the so-called allergic poison, causes this remarkable constriction of the involuntary muscles of the bronchi, and that is what brings on the attack of asthma.

In my experience I have observed probably the best results in the treatment of bacterial asthma. Of course, those cases in which the asthma is known to be due to a feather pillow or to a cat in the house are greatly improved with the removal of these agents; but very frequently we can do a great deal for the treatment of the bacterial type by the administration of the vaccine. I was interested to learn of Dr. Wilmer's experience with the stock allergins. My experience agrees with his, that we get very little information of value by the use of stock allergins. My practise is always to play out the patient's sputum and blood in order to make every attempt to secure staphylococci. I do not practise what Dr. Wilmer stated in regard to mixed vaccines. It is my custom to prepare a separate vaccine of each germ and then to test out those separate vaccines on the patient by means of intracutaneous tests.

Dr. Wilmer did not express his opinion on the relative merits of the scratch or cutaneous and the intracutaneous tests, but I would say that, as far as my experience is concerned, the intracutaneous test has proven the more satisfactory of the two, and then at the end of forty-eight hours we very soon learn which germ or germs from the sputum the patient is sensitive to, and these are the ones that are incoporated in a vaccine for treatment.

Now the relief from asthma is frequently very striking in these instances

of bacterial asthma, but I want to strongly endorse what Dr. Wilmer stated about the tendency to recurrence. It is always necessary to repeat a series of injections by the end of the first year. I know of several cases where this was not done, and with the return of the asthma. In other words, it is necessary to continue the administration of the vaccine for long periods of time.

It may also be stated that in bacterial asthma the vaccine very frequently has a similar beneficial effect upon the accompanying bronchitis, but not infrequently in the aged we have asthma, bronchitis, emphysema and bronchiectasis as a result of the long period. In these cases we can frequently relieve the asthma, but we are less successful in the treatment of the accompanying bronchitis, I think possibly because of the chronic anatomical changes that have taken place in the tissues.

I hope Dr. Wilmer will also discuss the allergic nature of the vernal catarrh or perennial rhinitis, cases in which there is a steady discharge of clear mucus from the nose. I have reason to believe that they are likewise allergic. I am sure the rhinologists here know to what I am referring—individuals of a sort of neurotic temperament, who without any apparent cause have this flow of pure discharge from the nose. I am sure we will appreciate Dr. Wilmer's opinion of cases of this sort.

Dr. S. Leopold: I am particularly glad to be asked to take part in this discussion, because it gives me an opportunity to express to Dr. Wilmer my appreciation of his very excellent résumé of the present state of our knowledge of this rather interesting and intricate subject.

Dr. Wilmer states in the first portion of his paper that many of these cases which present themselves to the internist for treatment have passed through the hands of the nose and throat specialist, and that I think is a common experience. I believe that possibly the majority of cases of asthma that I have seen—and I also include in that category cases of allergic vasomotor rhinitis, hayfever, etc.—have been operated on at some time or other in the hope of obtaining relief through the efforts of the nasal surgeon.

Then it seems to me we might consider the types of nasal pathology which are associated with asthma. The first type is the typical hayfever nose, in which the mucous membrane is characteristically pale, swollen edematous, and in which there is contact often between the turbinates and the septum. These cases are very frequently of an allergic type. They are sometimes relieved by the mere discarding of a pillow and sometimes by discontinuing the use of a tooth-powder containing orris. It therefore behooves the specialist for nose and throat work to keep in mind this type of allergic mucous membrane, because those cases are

best treated apparently by diagnosing them by means of the cutaneous test.

There is another type of nasal pathology which, in my experience, is associated with asthma, and which proper medication has completely relieved. I mean those that are characterized by the designation "bacterial asthma." I personally feel that as a diagnostic group it is the junk pile for cases that we cannot otherwise diagnose. At any rate, these cases, not ellergic in type, in middle life very often are associated either with enlarged turbinates, the posterior portions of which are in contact with the septum, or with sinus infection or polypi or other nasal pathology. I could quote certain ones which by being operated upon have been entirely and completely relieved of the asthma.

I am sorry that Dr. Wilmer did not take up the question of dust asthma. It is a very complicated subject, and certainly those of us who are seeing a great deal of asthma are struck by the number of cases which react to house dust. I personally believe that house dust is a conglomeration of various things, and any given dust may contain any of the animal substances which may act on a certain individual. If that substance from the wall of which Dr. Wilmer spoke had been inadvertently collected in the dust in the room, the case might have been diagnosed as a dust asthma. There is a protein-containing glue which is used in sizing plaster walls which has been found capable of producing reactions, and which in one case, reported by Cooke, showed that Le Page's glue produced a fatal anaphylactic shock by an intracutaneous test. I would like to know if Dr. Wilmer has used this substance in that particular case.

It is also a fact that cattle hair is used in plaster very largely, and it is just conceivable that in this rather remarkable case which Dr. Wilmer has cited, cattle hair might have been a factor.

I do not entirely agree with Dr. Wilmer in his classification as regards certain subdivisions of this subject. The atmospheric type of asthma I have been unable to substantiate in my work. Scheppegrell, who is probably the father of the study of hayfever in this country, and who has done most of the experimental work in isolating pollen, has very ingeniously constructed an apparatus which will alter the velocity of wind. His apparatus will produce a current of air going a certain number of miles per hour. By exposing gelatine plates at various distances from it, he is able to tell the concentration at a particular change in wind velocity. He has correlated this with the progress of hayfever cases in any given season. It is common experience for us here in Philadelphia to find that a hayfever patient going to the seashore suffers very much with a land breeze blowing and very little when there is a sea breeze. I think these cases classified as the atmospheric type by Dr. Wilmer, and in which

he says he has never gotten positive reactions, are simply cases of the inhalation type. Maybe it depends on the concentration of the doses which the patient gets. I have never seen a case of hayfever and asthma where the asthma has developed the first day of the hayfever. There, again, I believe it is a matter of concentration.

Just a word about the bacterial cases. I said that the classification of the bacterial cases was what I regarded as the junk pile in our diagnostic procedure. The cases that do not react to allergins are classified as belonging to the bacterial type. I do not personally believe that bacterial asthma is a definitely specific thing. I have had allergins made from the patient's sputum and have not succeeded in getting a cutaneous injection with the bacterial protein. I have had a bacterial protein made by the same method with which the animal proteins are made.

With these exceptions as to the classification of this type of asthma, and the fact that in my experience the dust cases which I grant are probably a group classification, my experience has been very much in accord with that of Dr. Wilmer, and I can add nothing further to his excellent presentation of the subject.

Dr. Wilmer (closing): In answer to Dr. Kolmer about the one thing he said with reference to the staphylococcus in the preparation of these vaccines, I agree with him on that. If I got a Friedlander or D. catarrhalis alone in a smear, I would discard it and go after more bacteria. I must get a staphylococcus before I consider that a vaccine is worth anything.

As to the value of intracutaneous and cutaneous reaction, I have not had a great deal of experience with intracutaneous testing, the reason for that being that I worked with Dr. Walker several times and we tried it out and he had a vast amount of material to work upon; but we had unfortunate results. In hypersensitive skins my test was very misleading. I have practically confined myself to the cutaneous type.

As to perennial rhinitis, I believe personally that a perennial rhinitis is a forerunner of a sensitiveness to some protein of some kind, and that if the patient should at any time become thoroughly impregnated with any particular protein, he would immediately break down and classify himself as a form of animal or hayfever.

As to Dr. Leopold's question about the glue, I have had no experience in that line. My patient got so well after the wall was painted that I did not go into the glue situation.

I agree with Dr. Leopold absolutely in his criticism of my atmospheric type. It happened that I have had some few cases who did not develop their asthma until after the wind storm had passed. Of course, I realize that the pollen was in the air, but the thunder definitely precipitated that attack, and it happens that I have had 2 cases recently quoted to me in which the same story was told.

Rhinoplasty by the Peak-roof Method

BY G. M. DORRANCE, M.D.

In planning for reconstruction of the nose there are four essential points to be decided upon:

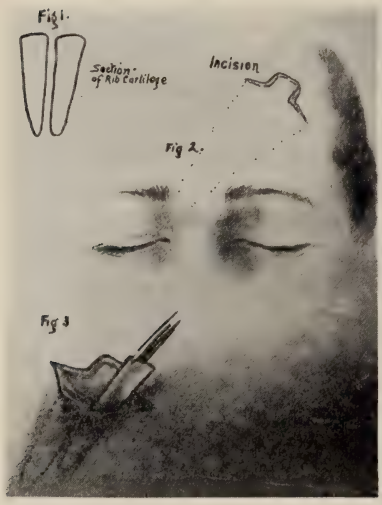
1. Where are the cutaneous flaps to be obtained?
2. What supporting material is to be employed?
3. How is the support to be constructed?
4. Is the nose to have a skin lining or not?

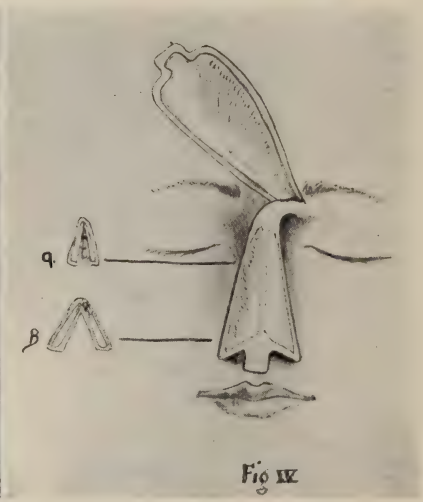
Whenever possible the tissue should be obtained from the forehead. The base of the flap is best placed at the inner angle of the orbit, so as to obtain the blood supply from the angular artery. In cases where a partial reconstruction of the cheek is necessary then the flap for the nose should be obtained from the temporal region, so as to utilize the pedicle for the closure of the defect in the cheek. Forehead tissue unquestionably forms the best nose, because the tissue is more rigid, has better color and does not have the tendency that other tissues have of taking on fat deposits. In addition to these reasons, we have a better blood supply, and the defects in the forehead can be more readily closed without leaving a disfiguring scar. The supporting material should be of cartilage. Bone has been successfully used, but has a tendency to undergo absorption unless it is in contact with other bone. Foreign materials, such as ivory, dead bone, or metal like gold or platinum, have all been tried with marked uniformity of failure. I wish to strongly advise against their use. In the Queen's Hospital at Sidcup, England, every conceivable form of support was tried. The consensus of opinion was that cartilage is superior to all others. This conclusion has been reached by careful observers in plastic surgery in every part of the world.

My own experience with cartilage has been very satisfactory In specimens of cartilage removed one, two and three years after its introduction reports from the pathologist indicate the cartilage to be normal in every case.

The problem next confronting us is, How are we going to obtain with our cartilage a support which will prevent the tip of the nose from drooping.

The method usually employed is to use one or two pieces of cartilage placed between the skin and the pericranium. When the flap is turned down to form the nose there is always a tendency for the tip to drop down. Gillies, recognizing this, placed the cartilage in the skin at the root of the nose, then turned this down before bringing the covering from the forehead. Neverthe-





less, the falling tip is a frequent complication. A careful study of the anatomy and bony structures of the normal nose convinced me that two lateral pieces of cartilage, placed as shown in the diagram, would give me the desired support. The cartilages are obtained from the eighth costal cartilage, and so trimmed as to be very narrow on the inner border with gradual thickening toward the outer edge. At the outer extremity of the future flap on the forehead an incision is made down to the pericranium, as shown in Fig. 2. The cartilage is inserted into the cavities made between the skin and the pericranium, as shown in Fig. 3. After

the cartilage has been implanted the edges of the incision are closed by suture.

You will note that between the cavities holding the cartilage a separation of 0.5 cc is obtained—in other words, the cavities are not connected and there is no tendency for the cartilages then to overlap. Six to eight weeks after the introduction of the cartilage the forehead flap is raised. As shown in the diagram the pericranium is included in the flap. After the flap is raised the edges of the pericranium are sutured together for one-half the length of the flap. This not only prevents the spreading of the edges, but avoids the necessity of lining the nose at this portion. The lower one-third may be lined with a free Olier-Thiersch graft or with skin from the surrounding area. The forehead wound can be closed by undermining the edges.

I refer to this method as the peak-roof method. I feel that it fulfils all the essential points of a normal nose excepting the septum. I have not found it necessary to replace the septum of the nose.

APRIL 18

Some of the Sources of Error in our Present Methods of Testing and Recording Hearing

BY GEORGE M. COATES, M.D.

Every otologist probably realizes the very great shortcomings of present-day methods of making hearing tests and recording results in such a form as to be intelligible to anyone reading reports or reviewing case histories. Indeed, the American Otological Society has an appointed committee at work on the standardization of hearing tests, but in its most recent report, that of 1922, it had only progressed so far as to state that efforts were being made to have tuning-forks of the most commonly used number of vibrations made as nearly standard in regard to tone

and length of sounding as possible. The Ninth International Otological Congress in 1912 adopted a method for the uniform recording of hearing-test findings which has not been put into universal application, but these two instances show that our branch of the profession of medicine has for a long time felt the need of some more uniform and exact method of ascertaining the degree of hearing a patient possesses and of recording such findings in a way that will be readily intelligible to others who may in turn repeat the tests with approximately the same results. Considerable experimental work has been done with this end in view, and from a talk with Dr. Fletcher, some time since, I understand that he will enlighten us tonight on the progress he has made in this direction. Those of you who have studied the *Transactions of the American Otological Society* and of the *American Laryngological, Rhinological and Otological Society* during the past few years will be familiar with the work of Dean and Bunch, of the University of Iowa, along this line with the tonal range audiometer and with the earlier work of the Western Electric Company presented last year before the latter Society.

The present brief paper is designed simply to place in concrete form, as an introduction to Dr. Fletcher's paper, the well-known defects of our present methods. The various tuning-fork tests usually employed, the Rinné, the Weber, the Schwabach and the Gelle, together with the modifications of the Galton whistle for testing the high tone limits, are all most useful, and, as a rule, suffice to make a diagnosis of the kind of hearing defect present; but they do not give us an accurate understanding of the amount of the defect. The much neglected Politzer test for tubal patulency will probably withstand even the more advanced methods of testing that seem about to open up to us. But for actual testing of hearing capacity the examiner is reduced to the employment of the voice (shouted, conversational and whispered) and to the watch or the acoumeter. In the use of these methods the personality and psychology of the examiner and of the examined play a large part as well as the method of examination employed.

The human voice, if it could be accurately regulated and standardized, should be the most useful instrument of examination, since it is the most familiar to the patient as well as what he most fears the loss of. Unfortunately it is the one test that cannot be standardized and the one where more errors of recording occur owing to differences in individual application. In the first place, when the loud voice, or even the conversational or whispered voice, is employed, it is almost impossible, in unilateral deafness, to exclude sound perception from the good ear. The usually employed methods of stopping the ear not under examination with the finger, either of the patient or the assistant, whether dry or moistened, by no means exclude perception of words even in ordinary tones, while the noise apparatus, which does exclude, also, by the very fact of the noise produced, diminishes sound perception in the ear under examination and also distracts the patient's attention. Again, the voice of the examiner cannot be standardized whether he speaks with a full or empty chest. Different examiners use different methods, and even the individual's voice varies from time to time, depending on catarrhal conditions in his respiratory passages, fatigue, etc. One examiner may have a sonorous bass and the next one a weak tenor or falsetto, so how can their results be expected to tally? The various elusive sounds of consonants or vowels may be used to a greater or less extent by different men or at different times, thus confusing results. Most examiners do not measure the actual distance at which the majority of spoken sounds can be repeated by the patient, but guess at or estimate it, which is a frequent source of error. Many consulting rooms are not large enough to get the full normal hearing distance, so that slight defects are unregistered. And lastly, no consulting rooms are sound-proof, so that extraneous noises must be reckoned with and their influence, perforce, estimated. Such extraneous noises vary greatly, depending upon the location of the office, the location of the room, whether back or front, on the street level or high above traffic noises, and also upon the time of year—whether the windows are closed or not. So it seems evident that voice-testing must be a very uncertain thing if accurate findings are desired.

The acoumeter test has the disadvantage that a large room is required, that the instrument may not be standardized, and that it is a test for a single sound, of fairly definite quality, to be sure, but not entirely familiar to the patient. It is conceivable that a patient may hear the acoumeter at a respectable distance and yet have very inadequate hearing for the voice and the various musical sounds of every-day life. The watch test is but a variation of the acoumeter test, or *vice versa*, and presents the same defects except that it is a sound the patient is accustomed to. Both tests are largely influenced by extraneous noises. The watch is unstandardized, but by a proper system of recording results may be made so to a degree. If the hearing distance of the watch used for examination is known by careful testing with a number of normal ears, this distance is used as the denominator of a fraction, say 50 inches. If the ear being tested gives us a hearing distance of but 25 inches, and this is used as the numerator, the resulting fraction is 25/50. If, now, another watch with a normal hearing distance of 40 inches is used the fraction should be 20/40. But the second watch may be of a lower or higher pitch than that first used, which may vitiate the result. The position in which the watch is held, whether in the palm or with the fingers, also affects the result. The watch test is, however, easily applied, quickly done and requires but little space, so that within its very obvious limitations it is not without a degree of usefulness.

Tuning-forks are used chiefly to determine the duration of perception, both by bone and by air, of sounds or musical notes of varying pitch. They are also used to measure the actual distance at which such sounds are heard by air conduction, and when so used the distance should be measured by a foot or metric rule and not estimated. This observation also applies to the watch. The defect in this method lies in the difference in habit of the person making the test. To be uniform, the fork must be of a standard number of vibrations and a standard size, and it must be struck with a standard degree of intensity to produce a standard sound. It must be used uniformly by either approaching or receding from the ear, but not by both procedures indiscriminately. It must

be held in the same position in relation to the examiner's hand and to the patient's ear.

All of these differences in practice must be reckoned with when the fork is used to detect diminution in air or bone conduction. If a stop-watch is used to secure exact data, much can be learned provided the fork is properly applied, is standard as to number and intensity of vibrations, being put into vibration by a standard degree of force. Necessarily, in this method, the duration of perceptible vibrations for the fork being used must be known definitely, so that any decrease from the normal may be noted. Fatigue and the psychology of the patient may make a wide divergence in results obtained, as many patients, particularly slow-witted or slow-thinking ones, seem unable to tell the precise moment at which the sound is lost. Vibrations felt instead of heard often confuse the issue. Neurasthenics and children are particularly at fault in this respect. The more commonly used method of comparing the time during which the vibrating fork is heard by the patient with the time during which it is heard by the normal ear is more simple and possibly less liable to error, provided the ear used for purposes of comparison is indeed normal. If the examiner's ear is used as a control, as is customary, repeated tests should be made at frequent intervals to determine this normalcy both for air and bone conduction, otherwise gross errors of diagnosis will be inevitable at times.

The Weber, Rinné, Gelle and Schwabach tests are not properly tests of auditory acuity, but are for purposes of diagnosis. Again, standardization of forks and methods of application are a necessity for the attainment of scientific results and again such standardization seems to be obtainable only within certain limits. For these tests, forks of a definite number of vibrations, preferably weighted to avoid confusing overtones, should be used, but, as Sonnenschein has pointed out, different forks and different applications of these tests are used by the most prominent otologists the world over. One man prefers the C^1 fork, another the C^2, while a third, equally distinguished, uses A forks exclusively. Presumably this makes little difference to the individual examiner, but it does make for a certain disparity in recording their results.

Sonnenschein notes, for instance, a variation in the place of application of the fork handle in making the Weber test. By some the fork handle is applied to the bridge of the nose, by others to the glabella with the head back to secure more uniformity in pressure, while the majority apply it on the vertex in the midline and occasionally it is applied to the chin or the teeth. In the Rinné and Schwabach tests the fork may be applied on the vertex or over the mastoid.

The principal common error obtained in testing for lowering of the high-tone limit is the lack of uniformity and accuracy in the instruments employed. The commercial Galton whistle is notoriously inaccurate and the patient often confuses the rushing air sound with the musical note to which it is desired he should pay attention. The Randall standardized whistle is much superior in this respect, as are the Bezold-Edleman whistles; but there still seems to be a possibility of error here. In addition to this, Randall has called attention to the fact that often apparent loss for high tones in the neurasthenic is psychological.

Isolated remnants of hearing in those profoundly deaf or in so-called deaf-mutes are very difficult to determine under present methods of testing, and yet, in out increased knowledge of what can be done toward reëducation in these cases, it is extremely important to know when these remnants exist. It takes a most elaborate series of forks, rods, whistles and other musical instruments to determine this point. If one instrument has been, or can be, devised that will give us a complete and comprehensive picture of upper and lower tone limits, duration of bone and air conduction for diagnostic purposes, acuity of hearing for ordinary sounds, and that will enable us to pick out isolated perception for single notes, the problem could certainly be considered solved.

Taken all in all, the instrument maker and the personal equation of both examiner and patient play such a large part in the commonly employed methods of hearing-testing that I feel sure the profession looks forward with the keenest interest to any device or method that will deliver it from at least some of these uncertainties and inaccuracies with which it has been struggling in the past.

Audiometric Measurements and Their Uses

BY HARVEY FLETCHER, PH.D.

RESEARCH LABORATORIES, AMERICAN TELEGRAPH AND TELEPHONE COMPANY AND WESTERN ELECTRIC COMPANY.

(*Notes on a Talk.*)

The talk comprised a discussion of the problem of making measurements of the degree of hearing and the practical applications of such measurements. It was illustrated by experiments with audiometers which were designed by the Western Electric Company in coöperation with Dr. E. P. Fowler.

Three types of audiometers were described and referred to as Types I, II and III. In each of these types the tone is delivered to the ear by means of a special telephone receiver. For the first two types the electrical generator supplying the current to this telephone receiver is of the audion bulb type. The various frequencies are obtained by varying the constants of the electrical circuit. In the third type of audiometer the generator is of the vibrating reed type. It gives only a compound tone of variable intensity. An analysis of this tone shows that it has frequency components throughout the important range of speech frequencies. The scale is arranged so that when the intensity dial is varied, until the threshold of hearing is reached, its reading gives the per cent hearing for speech.

The first type of audiometer was designed primarily for use in hospitals, medical schools and for highly specialized workers. It has a frequency range divided into twenty steps from 64[1] to 16,384 double vibrations per second, and an intensity range of approximately 100,000,000,000 divided into twenty-eight steps.

In order to demonstrate the performance of this audiometer the head receivers which are ordinarily used were replaced by a loud-speaking receiver so that all in the room could hear the

[1] In a redesigned audiometer this range has been extended to thirty-two vibrations per second.

various tones that the instrument produced. When high frequencies are produced in an ordinary room standing waves occur, due to the reflection from the walls. As a consequence of this it is extremely difficult to avoid errors in measuring hearing by the usual methods. Those in the audience verified the fact that moving the head through only small distances was sufficient to change the loudness of the sound from almost zero to very high values. This source of error is avoided when the sound is delivered directly to the ear by a telephone receiver.

The scales on the audiometers are arranged so that equal steps produce equal loudness changes as perceived by the ear If the intensity dial is rotated uniformly the tone dies away in the same manner as that from a vibrating tuning-fork held in the air. The great advantage of the audiometer over tuning-forks is that the tone can be held at a constant intensity at any desired value, whereas with the tuning-fork the intensity continually decreases. Also the intensity of the sound from the audiometer is definitely known in absolute units, so that if desired the actual amplitude of the sound wave in the auditory meatus can be calculated.

Type II audiometer is similar in performance to Type I, but is limited in its frequency and intensity range. Its frequency range is from 64 to 8192 double vibrations per second, being divided into eight steps, each step corresponding to an octave on a musical scale. Its intensity range is approximately 10,000-000,000.

As indicated above, the Type III audiometer is arranged to read directly the average per cent hearing. The zero hearing-point is taken as the average intensity where one feels the sound wave. The 100 per cent hearing-point is taken as the average threshold intensity for people who are known to have normal hearing. The first intensity-point corresponds to that produced when one talks very loud directly into the ear. The range of intensities between these two points is divided into one hundred equal loudness steps. This type of audiometer was designed to

fulfil the demand for a quick test and is particularly adapted for use in the army and navy, public schools and large industrial concerns.

By means of an audiometer similar to those described, an accurate measurement was made of the range of hearing in intensity and frequency for persons having normal hearing. The final results of such measurements are shown in Fig. 1. The abscissæ represent frequencies which are expressed both directly and logarithmically along the axis; the ordinates represent intensities

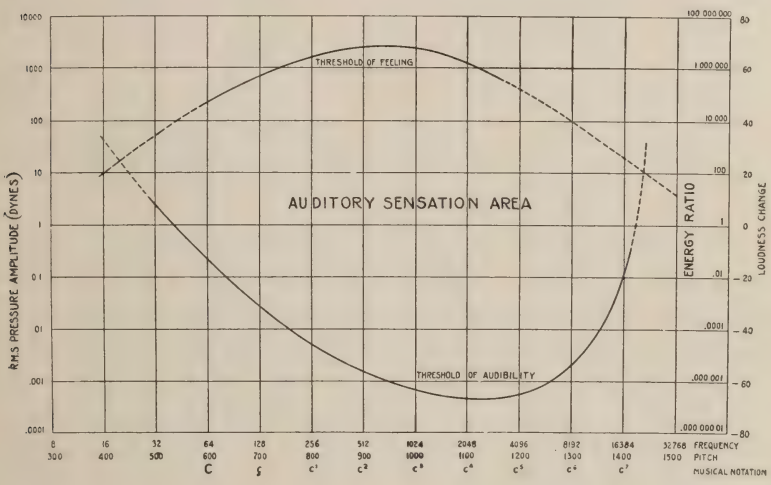


FIG. 1

and are expressed both as amplitudes of pressure variation (of the sound in the auditory meatus) and as loudnesses in loudness units.[1] The lower curve gives the threshold of hearing; the upper curve the threshold of feeling. Their intersection points are at the upper and lower limits of audibility.[2] The area enclosed between these two curves has been named "the auditory sensation

[1] For discussion of loudness units, see paper given before the Franklin Institute March 29, 1923, published in the Journal of that Institute.

[2] For methods of determining these curves, see papers by H. Fletcher and R. L. Wegel in the Physical Review, June, 1922, No. 6, xix, and Proceedings of the National Academy of Science, July, 1922, No. 7, viii.

area." Each point in it corresponds to a particular tone having a definite frequency and intensity, which can be sensed by the average ear. The pitch and loudness scales are so chosen that the numerical value of the area of any portion of this auditory sensation area is approximately equal to the number of pure tones in this region that are perceptibly different.

Typical audiograms or threshold-of-audibility curves for six types of deafness are shown in Fig. 2. The threshold of hearing-curve is represented in its relation to the normal auditory-sensation area. The fraction of the normal auditory area above the abnormal threshold curve is taken as the per cent hearing, since it represents approximately the number of pure tones available for the deaf man's use compared with those available for the average person of normal hearing. The Type III audiometer is designed to give very quickly an approximate value of the per cent of hearing as thus defined.

For clinical purposes one is usually more interested in departures from the normal than in absolute values. In Fig. 3 the same audiograms are shown as those in Fig. 2, but they are plotted as departures from the normal, *e. g.*, we plot the distance from the normal to the abnormal curve measured in loudness units as a function of frequency. The Type II audiometer is designed so that such curves can be plotted directly from the scale readings on the intensity dial.

There are three principal uses for audiometers, namely:

1. For making physical examinations of candidates for schools, for the army and navy, or for large industrial concerns, etc.

2. For diagnostic purposes.

3. For obtaining data for prescribing aids to the hearing. In school it is very desirable to separate those with abnormal hearing from the other children, so that they may receive special instruction. Also, the pupils in this segregated group should again be divided into probably three or four groups according to their degree of hearing. This can be done very quickly with the Type III audiometer.

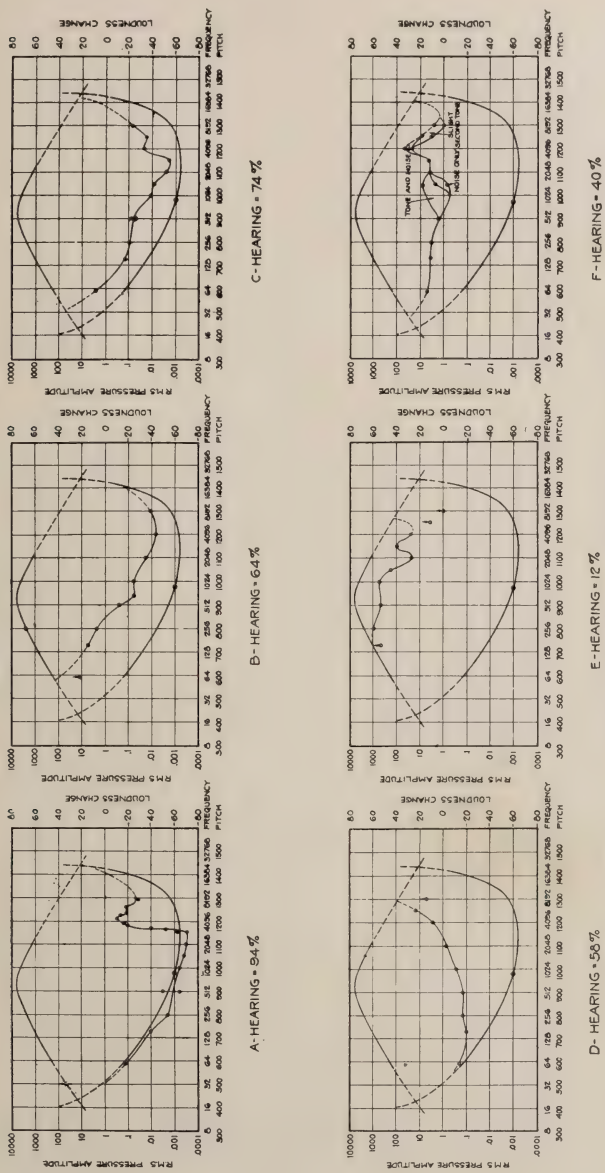


Fig. 2.—Audeograms for Typical Cases of Deafness.

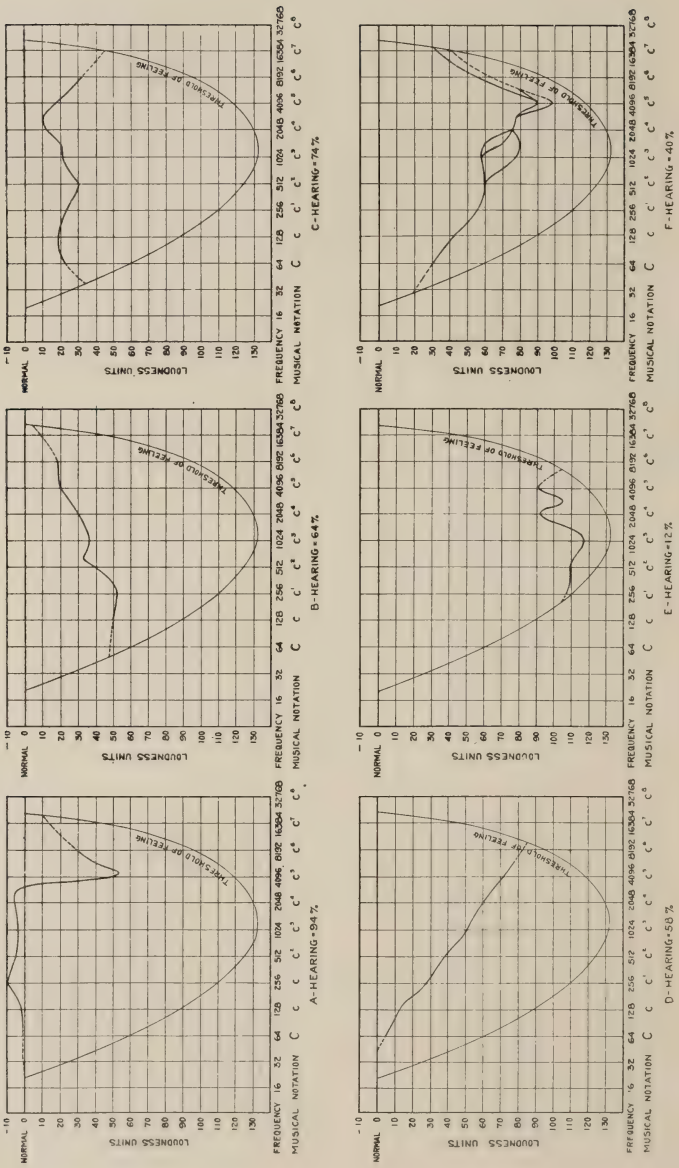


Fig. 3.—Audeograms for Typical Cases of Deafness.

The usefulness of audiometers in the army and navy is very evident. They will be extensively used not only for measurements on applicants but for periodic measurements of those in the service.

The full significance of audiometric measurements for diagnostic purposes is not at present understood. A full understanding can come only after the ear specialists have had considerable experience with these instruments. However, it seems reasonable to expect that when accurate audiograms for each known case are kept on file, information will soon accumulate from which some important generalizations can be made. Recent discussions of this phase of the work have been made by Dean and Bunch,[1] Wilson and Minton[2] and Fowler and Wegel.[3]

The third use mentioned has received little attention by ear specialists. This is due principally to two facts, namely: (1) That the physical properties, expressed in definite numerical terms, of the existing types of aids to the hearing are not generally known; (2) the existing audiometric measurements are not sufficiently standardized to enable an otologist to give accurate numerical figures for the degree of deafness. There are a number of factors which must be taken into account before one can tell accurately whether any of the existing sets will materially aid the hearing, but a few general principles will be set down here to serve as a beginning.

The proper volume of speech for any case of deafness can be determined rather accurately, but it will require considerable experience with the various types of deafness before one can expect great proficiency in prescribing deaf sets. For the purpose of determining the proper volume of speech necessary for good hearing a set of curves showing the relation between the interpretation and the volume or loudness of speech was determined. The quantitative measure for this interpretation is taken

[1] Audiometer in the Otological Clinic, Am. Otol. Soc. Proc., 1920, Part II, vol. xv: Laryngoscope, August, 1919.

[2] Proc. Inst. Med. of Chicago, 1921, pp. 157–171.

[3] Audiometric Methods and Their Applications, Trans. Am. Laryn., Rhinol. and Otol. Soc., 1922.

as the per cent of received syllables which are properly understood and is called "the articulation." Such a set of curves is shown in Fig. 4. These curves were determined by a large number of tests[1] with persons of normal hearing and from our limited experience with people having abnormal hearing. The curve marked "100 per cent hearing" shows the articulation for persons of normal hearing as a function of the "loudness" or "volume" of speech, which is plotted along the axis of abscissæ

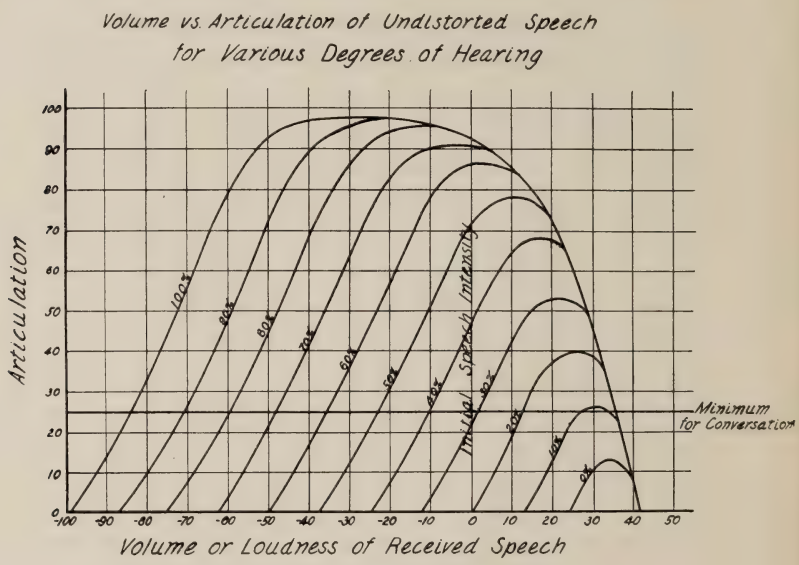


FIG. 4

and with the zero at the loudness corresponding to a speaker talking directly into the ear with normal conversational volume. The intensity corresponding to this standard loudness is called "the initial speech intensity." It is seen that this curve intersects the axis of abscissæ at 100. This shows that speech can be diminished 100 loudness units below its initial intensity before

[1] Fletcher, H.: The Nature of Speech and Its Interpretation, Franklin Institute, June, 1922.

it becomes inaudible. This number of loudness units corresponds to an intensity ratio of 10^{10} or 10,000,000,000.[1]

The best loudness for interpreting speech is 30 units below the standard loudness as defined above. At 60 units below this standard one still obtains good interpretation if his hearing is normal and if there is no noise present. At 90 units below this intensity one can scarcely hear anything but the loud vowel sounds. The falling off of the interpretation at high volumes is partly due to the distortion produced by the ear on account of the large number of subjective tones which are introduced at these high intensities. The introduction of these subjective tones makes it difficult to sense the speech sounds in their proper proportions.

The other curves in this figure give similar relations for persons having various degrees of hearing. In these curves the threshold of audibility for speech, which is also the zero articulation point, is shifted toward the standard loudness or zero point on the axis of abscissæ. This shifting is accompanied by an increased ear distortion. The amount of this ear distortion varies greatly with the type of deafness. These curves give the probable values of the articulation for various degrees of deafness of the kind in which there is not greater distortion than in the normal ear.

To find the articulation on the per cent of speech syllables which are correctly interpreted for a deaf person having more ear distortion than normal, the values from these curves must be multiplied by a factor which we will call "the deafness distortion factor." This factor is dependent upon the type of deafness and can be determined experimentally for each case. This is done by pronouncing directly into the ear (lips less than one-half inch away from the ear) a list of monosyllabic words and finding how many the patient interprets correctly. In this test care should be taken not to raise the voice above the normal conversational volume. From the set of curves in Fig. 4, read the articulation corresponding to the initial speech intensity on the

[1] Fletcher, H.: Physical Measurements of Audition and Their Bearing Upon the Theory of Hearing, Journal of Franklin Institute, July, 1923.

particular curve corresponding to the per cent of hearing obtained from the audiometric measurements. The ear-distortion factor is then found by dividing the per cent of words interpreted correctly under the conditions described above by the value of this particular ordinate. For cases having more than 50 per cent hearing it is more convenient to make the articulation test with the mouth one foot away from the ear. When made in this way the ordinate at -23 instead of at 0 should be used in obtaining the ear-distortion factor.

With this data available it is possible to determine approximately the articulation that will be obtained by the patient at any volume of received speech. For example, let us assume that the audiometer tests indicate that the patient has 40 per cent hearing and the articulation measurements at the standard loudness show 30 words out of 100 are correctly interpreted. The ordinate of the curve at abscissa zero for 40 per cent hearing is seen to be 47 per cent. Then the ear distortion factor is $\frac{30}{47}$, or 0.64. By talking very loudly and directly into the patient's ear a loudness of approximately 10 units higher than standard can be attained. With this loudness the 40 per cent hearing curve gives 70 per cent articulation which when multiplied by 0.64 gives 45 per cent. This then is the approximate articulation that the patient would obtain under such conditions. For certain types of deafness this factor is so small that no amount of amplification of speech would enable the patient to interpret enough syllables to understand satisfactorily. However, it must be remembered that a low distortion factor may be due to a lack of education because the speech sounds are heard so infrequently. With a deaf set giving the proper volume this distortion factor should increase very materially with practice. From a very limited experience we have drawn the line at 25 per cent articulation as representing the least syllable articulation that one can have and still be able to carry on a conversation.

In Fig. 5 curves are shown which give the volume efficiency of typical hearing aids now on the market. The ordinate gives the loudness of the speech at the ear measured on the usual scale when

the speaker is at the distance from the transmitter (or in the case of the curve marked "average speech unamplified" the distance from the ear) given by the abscissa. This curve represents the effects in a room 25 x 18 feet. From the curves in Figs. 4 and 5

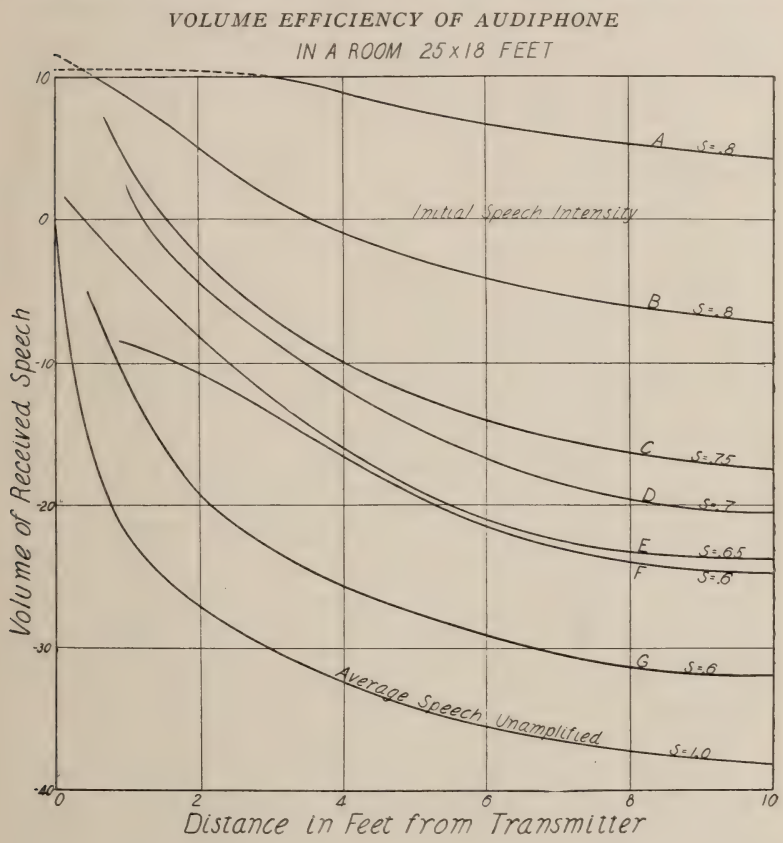


FIG. 5

it will be seen that for the case of deafness considered above namely, 40 per cent hearing, the patient could not hear speech at distances greater than a foot and a half from the ear if it were spoken at the average conversational volume.

These sets of curves enable one to prescribe the proper type of set from a volume standpoint. For example, consider the case just mentioned. For an ear distortion of 0.64 and with a deaf set, which gives perfect reproduction, the volume which must be delivered in order to keep the per cent articulation above 25 must be −6 units. The ordinate corresponding to this volume in Fig. 4 is 39, which when multiplied by 0.64 gives 25 per cent. Deaf set "C" would give the required volume for distances less than two feet, deaf set "B" would be satisfactory for about five feet, and deaf set "A" would enable the patient to hear the speaker anywhere in a room even larger than that in which these tests were made.

These conclusions are correct only if the deaf set gives perfect reproduction for speech. Of course, this is far from being the case. Another factor must be introduced which we will call "the deaf-set distortion factor." Such factors are given on the curves in Fig. 4 and designated by the letter "S." This factor has been determined for the case when the speaker is three feet from the transmitter. It was obtained by finding the articulation of speech delivered by these sets to one having normal hearing. For smaller distances this factor is usually greater and for larger distances less than that for the three-feet distance, the amount depending on the acoustics of the room. Some account can be taken of these acoustic effects, but I do not desire to enter into a detailed discussion of that phase of the subject here.

For the case of deafness considered above the set "B" used at three feet distance would then give an articulation of 55 per cent $\times$ 0.64 $\times$ 0.8 = 28 per cent. The set would then be sufficiently satisfactory to carry on a conversation at this distance, although with some difficulty. If the deaf set "A" were used the articulation would be 70 $\times$ 0.64 $\times$ 0.8 = 36 per cent at three feet distance, which indicates a satisfactory interpretation of speech. Even at a distance as great as ten feet the articulation would be satisfactory.

This method of combining the ear distortion factor and the deaf-set distortion factor, that is by multiplying one by the other,

gives results which agree approximately with those observed with persons having average types of deafness. However, it is expected that further research will reveal a method of correcting the deaf set so that the distortion factor of the set will be very different for different types of deafness. For such cases it is expected that the simple method of combining these two factors which has been described here will not apply.

It will be seen from the curves of Fig. 4 that when one has 0 per cent hearing, no known aids will enable him to understand speech satisfactorily. When speech is amplified the energy of the vowel sounds is sufficient to cause annoyance and even pain before the volume of the consonant endings are sufficiently loud to be heard. It is by virtue of these endings that speech is interpreted, so although 0 per cent hearing, as used here, does not necessarily imply that the person cannot hear any sounds, it does mean that the auditory sense cannot be used to interpret speech even when aided by the best means known. Even with 20 per cent deafness it is very unlikely that a deaf set will give any satisfactory help, although if in such a case it happens that the ear distortion is very small the type "A" deaf set may give some aid.

This method of predicting the amount of aid received from using various types of deaf sets is admittedly only very approximate, but, nevertheless, it should be very useful. Our experience with a limited number of cases of deafness has shown that it agrees approximately with the observed results.

It is hoped that this preliminary discussion of so important a matter may stimulate sufficient interest to cause some workers in the otological field to take a serious interest in the problem so that in the near future we may be able to answer satisfactorily the questions so important to one suffering from loss of hearing, "What type of apparatus will aid my hearing and how much improvement should I expect by using such a device?"

MAY 18

Plea for an International Investigation into Otosclerosis and Allied Forms of Deafness

BY J. S. FRASER, M.B., C.N.B., F.R.C.S. (EDIN.).

I want to say, first of all, that I appreciate very highly indeed the honor you have done me in asking me to address you. I hope you will excuse me if, in what I am going to say, I lay special stress on the work—unfortunately too meager—which has been done on otosclerosis in Great Britain.

Otosclerosis has been for long the opprobrium of otology. I have nothing startling, or even new, to say about this affection. You will all agree that our knowledge of otosclerosis is in a very vague and unsatisfactory condition. It is for this reason that I venture to suggest the need for an international investigation into the subject. If this can be arranged, I believe that it will repay otologists for their time and trouble.

It is a very sad thing to be confronted with a case of deafness, with or without marked tinnitus, in a young or comparatively young woman, and to have to confess that in the present state of our knowledge little or nothing can be done to relieve her. I remember Dozent Dr. (now Prof.) Neumann, telling me that when he visited the United States he was sometimes rung up and asked to see a patient in consultation. He asked: "Is the patient a young woman with deafness and tinnitus? If so I am not coming. I know what the case is already—otosclerosis."

At the present time otologists spend many hours in making a diagnosis by means of a tuning-fork and other tests between two kinds of non-suppurative deafness, nerve-deafness and otosclerosis, for neither of which they can do much. I often feel that these tests are rather a waste of time.

Everyone will admit that the present position is unsatisfactory. What can be done to alter it?

You will remember that shortly before the war an international investigation was commenced into the subject of ozena. Undoubtedly ozena is a very unpleasant disease which interferes with or destroys the sense of smell. Still, hardly anyone will claim for it a place of importance equal to that occupied by otosclerosis. This country is the home of "team work," as applied to the investigation of individual cases. Why should it not take the lead in advocating the creation of an international team to elucidate the subject of otosclerosis?

It may be objected that the present is not a suitable time for such an inquiry. I reply that science is international and that scientists may well set an example.

Etiology. 1. *Sex.* Why is otosclerosis so common in the female sex? On looking over my records of private cases during the last eighteen years (1905 to 1922), I find that out of 255 cases 183 were female and only 72 were male patients. From the case records of the Royal Infirmary, Edinburgh, belonging to Dr. Logan Turner and myself, I see that from 1914 to 1922, inclusive, we had 458 cases, of which 354 were female and only 104 male.

2. *Heredity.* It is stated that a family history of deafness can be obtained in from 30 to 80 per cent of cases; usually about 50 per cent. Gray thinks that the percentage should really be higher than 50, because, as he puts it, it appears to be an article of faith with some individuals that no qualities are inherited in their own particular families except those which are desirable. We must never accept the patient's statement that there is no deafness in the family. Gray holds that the primary fault is an inherited defect in the organ of hearing and that all other general and local conditions are merely contributory. As regards otosclerosis, Gray looks on the human race as a series in a scale. At one end we find individuals in whom there is almost no potentiality for the development of otosclerosis; at the other end are those in whom the potentiality is exceedingly strong and in whom almost no special stimulus is necessary to produce the disease. Gray believes that otosclerosis is a variation and that such variations are more apt to occur in organs such as the cochlea, which are of

comparatively recent origin, than in those like the vestibular apparatus, which are of more ancient descent. For this reason the margin of the oval window, the foot-plate of the stapes, and in some cases the nervous structure of the cochlea are affected, while the other parts of the labyrinth are untouched. Yearsley believes that otosclerosis is transmitted in the female line, but this is doubtful. Körner has suggested that otosclerotics should not marry, but this seems to me rather a "large order."

3. *Congenital or Developmental Deafness and Otosclerosis.* Cases of congenital deaf-mutism have been examined by Politzer, Habermann, Schwabach, Panse, Denker, Alexander, Lindt and Nager, in which a focus of otosclerosis was found in the labyrinth capsule. The connection, if any, between the two conditions is by no means clear. Alexander believes that there is a stepping-stone between congenital deafness and the various forms of congenital hardness of hearing, that the first rudiments of otosclerosis are often of congenital origin and that the ankylosis of the stapes and atrophy of the sensory epithelium are secondary. These congenital foci may exist without giving rise to symptoms until the age of puberty. Körner believes that otosclerosis is due to certain determinants in the germinal cells of the parents, but is influenced by puberty, parturition, the climacteric period and also by diseases of the middle ear. He explains cases of otosclerosis with no apparent heredity by supposing that the disease has skipped some generations. Hagener believes that the primary condition in otosclerosis is a change in the eighth nerve, and, in support of this, points to cases in which tinnitus is the first symptom of the disease. Manasse has shown that the nerve changes are the same in advanced otosclerosis and in pure nerve deafness. We know that there are cases of atypical otosclerosis in which functional examination apparently shows pure deafness, and yet, on microscopical examination, a typical focus is found in the labyrinth capsule. Such cases have been recorded by Alexander, Kalenda and Stern. This form of the disease is markedly hereditary. Manasse finds that in otosclerosis there is little or no connection between the changes in the labyrinth capsule and the

results of clinical examination. Even with a small bony focus we may have the clinical signs of otosclerosis, and, on microscopical examination, the well-marked picture of nerve deafness. Indeed, in one case there was in the better-hearing ear a focus of otosclerosis, while on the other side the labyrinth capsule was normal. Goerke apparently believes that the focus of pathological bone is only incidental and not of great importance. Nager, however, holds that if the otosclerotic focus adjoins the stapediovestibular ligament we have the well-known Bezold's triad of signs.

Stein is of opinion that in otosclerotics there are always other diseases of degenerative origin. Gray regards otosclerosis as a degenerative process and suggests still another name for the disease, "idiopathic degenerative deafness." Hammerschlag notes that the bony changes characteristic of otosclerosis have been found in many cases of congenital deafness, not only in the hereditary degenerative (sporadic) type but also in endemic cases. He reminds us that there are families in which cases both of hereditary deafness and otosclerosis are to be found. Indeed, he regards otosclerosis, progressive nerve deafness and hereditary deaf-mutism as offering manifestations of the same hereditary disease. These facts suggest that hereditary deafness and otosclerosis are to be regarded as different forms of one and the same pathological process.

Edinger has suggested that hereditary deafness appearing in middle age belongs to the group of wasting diseases in which the normal demands of the organ cannot be replaced. Progressive nerve deafness is obviously to be included in this group, and Goerke wishes to put otosclerosis in the same category.

4. *Age.* The age incidence of otosclerosis is difficult to estimate. I find it very common in girls and young women between eighteen and twenty-six years. Probably slight deafness has been present for some years before the patients seek advice, so that it is likely that the real onset of deafness occurs about the age of puberty.

5. I need not enumerate all the various toxic and other conditions to which otosclerosis has been attributed. I may, however,

mention anemia, intestinal sepsis, pregnancy and the puerperium. For once syphilis does not seem to be a guilty party. The results of the Wassermann reaction and the greater frequency in the female sex are against syphilis.

6. A dystrophic change in the nerve supply of the region is said by some to be the primary factor in the production of otosclerosis. We know that Froeschels has described the tickling symptoms as absent or greatly reduced in cases of otosclerosis, but the value of this finding has been discounted, since it was also reported in other ear diseases. Further, it is also possible that the dystrophy may be due to the presence of the lesion.

7. Abnormalities of the endocrine glands have been advanced as the main factor in the production of otosclerosis. No doubt one appears very "up to date" nowadays when one attributes an obscure disorder, such as otosclerosis, to an affection of the ductless glands; but what proof have we of the connection, if any, between the two? The ductless glands are "all the rage" at present. It is true that changes in the labyrinth capsule similar to those seen in otosclerosis have occasionally been found in cretins and in patients suffering from goiter. In most cases, however, of endemic deafness and deaf-mutism no such changes were discovered. Italian otologists tell us that otosclerosis is common in cases of osteomalacia, a condition in which the ovaries and suprarenals are said to be involved. Recently, work has been done which tends to show that in otosclerosis the calcium content of the blood is diminished. Lastly, disorders of the pituitary and parathyroid glands are credited with producing pathological changes in the bony skeleton. Certainly, I have never seen any improvement in otosclerosis from the administration of extracts of the ductless glands, singly or in combination. The subject certainly calls for investigation, but I am afraid that at present the use of the words "disorder of the endocrine glands" is regarded by many as rather comforting, like that of the blessed word "Mesopotamia."

8. *Past Attacks of Otitis Media.* On this point opinions differ greatly, and it is very difficult to clear up the question. Almost

all children have had at one time or another an attack or attacks of earache, so that it is impossible to exclude otitis media as a cause of otosclerosis. The usual objection is that the drumheads are often normal, but does this exclude the possibility that such cases may be secondary to otitis media? I do not think so. Is it not possible that while the membrana tympani returns to normal the local infective process may linger in the region of the anterior margin of the oval window, a *locus minoris resistentiae?* On the other hand, even the supporters of the "otitis media" view—among whom I place myself—do not claim that inflammation of the middle ear explains everything. All cases of purulent rhinitis due to scarlet fever or measles do not end in ozena, nor does every case of bronchitis end in bronchiectasis. In the vast majority of cases otitis media passes off and leaves no deafness behind nor any change in the drumhead. This I have observed in my own children. Can we be quite sure, however, that in patients with an hereditary tendency to otosclerosis the inflammatory condition may not in time invade the bone? In certain families the auditory apparatus may be congenitally weak and therefore unable to throw off completely an attack of otitis media. Bryant states that he believes in the connection between nasopharyngeal catarrh and otosclerosis. Lucae in his book on *Chronic Progressive Deafness* stated that no sharp dividing line could be drawn between chronic middle-ear catarrh and otosclerosis.

Nowadays it is usual to divide cases of otosclerosis into primary and secondary, the latter being those which result from otitis media. Prof. Gustav Alexander put it to me thus: "We know that some cases of otosclerosis follow otitis media; the question is, Do all cases?"

9. *Congenital Weakness of the Mesoblastic Tissue.* The conjunction of otosclerosis with fragilitas ossium and blue sclerotics is of considerable interest. I have examined three such cases, all from one family. All three structures—the labyrinth capsule, the long bones and the sclerotics—arise from the mesenchyme.

10. *Primary Affection of the Labyrinth Capsule.* Many otologists look on otosclerosis as a disease which occurs primarily in the

bone of the labyrinth capsule, the infection coming by way of the blood stream as in osteomalacia, rheumatism, syphilis, etc. We must remember, however, that formerly tonsillitis was held to be of rheumatic origin, *i. e.*, that the tonsil was supposed to be infected through the blood stream. Now we believe that many cases of rheumatism are of tonsillar origin, *i. e.*, that the blood stream is infected from the tonsil. In the same way, in my opinion, the number of cases of otosclerosis which are regarded as secondary to otitis media is likely in the future to grow larger, while those looked upon as "primary" will probably decrease.

There is too great a tendency to attribute otosclerosis to one cause alone. Is it not possible that several causes may be combined? I hold that an attack of otitis media may be compared to the match or cigarette end that lights the fire. The hereditary tendency in the female sex corresponds to the "inflammable material. Any weakening of nerve influences and disorders of the ductless glands which preside over the processes of bone formation and repair may be likened to a want of water with which to extinguish the fire."

Pathology. The trouble in regard to the pathology of otosclerosis is that few general pathologists appear to know much about the diseases of bone and that few otologists can be regarded as expert pathologists. The labyrinth and its capsule are not easy tissues to prepare for microscopical examination. I had long wanted to obtain the opinions of eminent pathologists regarding the nature of the changes seen in otosclerosis when, in 1919, there was a meeting in Edinburgh of the Pathological Society of Great Britain. Though not a member, I was asked to contribute a paper, and I thought it would be a good opportunity to collect opinions. In order that the assembled professors and others might hear and see both sides of the question, I asked Dr. Gray, of Glasgow, to attend, so that we both might put our views before the meeting. Well, we showed our microscopical preparations, gave our own opinion of their interpretation and waited for the words of wisdom. The assembled pathologists showed a surprising unanimity: they said nothing. I got up again and explained

that the matter was a very important one, as the disease affected young and often very pretty girls. Even that failed to elicit any opinion as to the pathological nature of the affection.

We have only to remember the number of names given to the condition commonly known as otosclerosis to realize that there is no general agreement as to the pathology. I need not enumerate these titles, for they are well known to all of you.

Observers admit that the first stage is a spongification of the bone with the formation of large spaces containing a central blood-vessel surrounded by granulation or connective tissue. The bone forming the walls of this space stains deeply with basic stains such as hematoxylin. Some believed that this is new-formed bone, but, in my experience, new bone takes on the eosin stain. I believe that the deeply staining bone is merely the old bony tissue, the staining reaction of which has been altered by the change it has undergone owing to the vascular starvation and increased supply of inflammatory lymph.

Secondly, observers admit that the final stage is one of sclerosis or formation of dense, more or less irregular lamellar bone, which stains with eosin. Differences of opinion are revealed when we come to the presence of osteoclasts. Some hold that these cells are responsible for the removal of bone in the early stages of the disease, while others—probably the majority—hold that osteoclasts only appear at a more advanced stage. In the early period bone absorption is probably due to pressure of the new-formed granulation tissue. In any case osteoclasts are not necessary for bone absorption, as one sees from the examination of the labyrinth capsule in cases of tumor of the eighth nerve where the internal meatus is greatly dilated, and yet no osteoclasts can be found.

Undoubtedly the process of bone absorption and the formation of large vascular spaces appear to proceed along the bloodvessels which can be seen to enter the labyrinth capsule from the muco-periosteum in the anterior margin of the oval window.

Bony ankylosis of the stapes is probably a late feature of the disease. It was present in only 2 of Gray's 4 cases, and both of these were of many years' duration. In my early case it was

not present, but in the second case, an old woman, aged sixty-five years, it was marked on both sides.

Otosclerosis is not always confined to the site of election. The region of the bony prominences of the lateral canal may be involved as in my second case. Further, the affection may extend round the cochlear capsule between the lamellar and the cartilage bone to the region of the internal meatus. Politzer held that the presence of otosclerosis in this latter situation proved that the disease was not due to otitis media, as there was no mucosa present here. There is, however, a lymph or connective-tissue space between the cartilage and lamellar bone—well marked in the fetus and in young children, less evident in adult life—along which the chronic inflammatory process may spread.

As Katz has pointed out, change may occur in the ossicles in cases of otosclerosis similar to that seen in the labyrinth capsule. I have observed the same thing in the malleus and incus in cases of chronic middle-ear suppuration and cholesteatoma. We must remember that the tympanic ossicles and joints are almost the only bones and joints in the body which are covered merely by mucous membrane, and so liable to disease from the surface. We know how often inflammatory affections of the nose lead to bone disease of the turbinals, *e. g.*, cases of nasal polyps and ethmoiditis.

The anterior margin of the oval window is a weak spot. This may be due to the presence of the tensor tympani tendon or to the greater movement of the anterior part of the foot-plate of the stapes, as Breuhl suggests; but it appears more probable that its vulnerability is due to the entrance of vessels from the deep layer of the mucosa, associated with the presence here of fibrous tissue strands which replace cartilaginous "rests," well seen in the fetus and young subjects. This piece of cartilage is connected by a narrow strip to the anterior margin of the oval window. I have observed that this strip divides like a Y, one end becoming continuous with the endosteum of the labyrinth, and anterior to the stapes foot-plate, while the other joins the mucoperiosteum of the window niche. Mayer states that the island of cartilage

may remain unossified until after the second year—indeed, ossification may never occur and the medullary space may remain filled with fatty masses. .Mayer points out that two centers of ossification meet at the site of election, one from above and the other from below. These findings lend support to the view of Neumann and others that otosclerosis is a congenital anomaly of the process of growth in the petrous pyramid, an anomaly which only gives rise to symptoms (deafness and tinnitus) after the age of puberty. The idea is that while the growth of the petrous bone should normally cease in childhood, in otosclerotics the growth goes on and results in the formation of vascular, spongy bone.

Mayer thinks that otosclerosis may be in the nature of tumor formation (hematoma). In two or three cases he found that otosclerosis was associated with the presence of a small neurophroma of the eighth nerve and thinks this fact significant. Many years before, Gray had likened the area of otosclerosis to an enucleable foreign body in the labyrinth capsule and had suggested that it might be a sort of a special necrosis, due to interference in the blood supply. Mayer also attributes importance to these circulatory changes, which may lead to reconstruction of the bone in the affected area.

Bryant has called attention to the similarity of the changes seen in otosclerosis to those observed in rickets, osteomyelitis, otitis fibrosa (v. Recklinghausen), osteomalacia, osteitis deformans (Paget), osteoarthritis and syphilitic bone disease. Mayer, Nager and Jenkins have all called attention to the change in the labyrinth capsule in the case of osteitis deformans. Lieberman has compared spongification of the labyrinth capsule to the change seen in a rib in empyema.

The question arises: Is otosclerosis a pathological entity? Are we examining different stages of the same disease or are we observing different diseases? Deafness, tinnitus and giddiness occur in cases of osteitis fibrosa, while on micro-examination exostoses and stapes ankylosis have been found (Lantern Demonstration).

Symptoms. Breuhl divides cases of chronic progressive deafness into:

1. Nerve deafness, *i. e.*, labyrinth atrophy without bony changes.
2. Labyrinth atrophy with spongification but without ankylosis.
3. Pure middle-ear deafness, *i. e.*, stapes ankylosis without atrophy.
4. Mixed middle and inner ear deafness, *i. e.*, stapes ankylosis with atrophy.

I will not weary you with a detailed account of the symptoms of otosclerosis, which are only too well known. In several cases I have found that tinnitus was present for a considerable period before deafness came on. Further, if nerve deafness is present in a female patient under forty years, and if the Wassermann reaction of the blood and cerebrospinal fluid be negative, I am very suspicious that the case is one of atypical otosclerosis, especially if there is a history of deafness in the family. Many of my cases of otosclerosis laid stress on the fact that they were worse during a cold. This is not to be wondered at, but one sees it mentioned as a point against the diagnosis of the disease. Deafness may be due to: (1) Stapes ankylosis, but this is a late feature. There may, however, be a certain stiffness of the oval window structures long before ankylosis occurs. This may be compared to the difficulty of sliding out and in the drawers of a badly made bureau. (2) The deafness may be caused by changes in the labyrinthine fluid, as suggested by Jenkins and others, a point difficult of settlement. (3) Nerve degeneration may obviously cause hardness of hearing, but this again is usually a late feature. In many cases with marked deafness for speech there are only the tuning-fork signs of obstructive deafness while the upper tone limit is normal and the watch well heard by bone-conduction. (4) I agree with those that hold that the spongification of the labyrinth capsule is the cause of deafness and tinnitus. Normally, the nerve endings in the labyrinth are surrounded by dense, almost avascular bone and the presence of spongy bone richly supplied with bloodvessels in the capsule of the cochlea is bound to interfere with hearing. As so many of the patients say, "I could hear well enough if it were not for the noises."

Lately two curious cases were reported by Gray in which the patients heard better during a cold in the head and an attack of hayfever respectively. Other two cases heard better when a draft of cold air was admitted to an overheated room. The second of these patients took a little brandy and then suddenly experienced a great improvement in hearing. I have been told of a case in which an old, deaf man suddenly heard very well just before death. It is said that the inhalation of amyl nitrite or chloroform or the administration of pilocarpin or a warm douche to the ear may cause sudden improvement in hearing. The explanation of these phenomena may be either in alteration of the labyrinthine tension or of the local circulation.

Paracousis. Politzer believed that paracousis was due to the vibration produced by the noise of machinery, for example, exercising a sort of massage effect on the ossicles and joints. On this view one would expect that the beneficial action would continue for some time after the noise had ceased, but as a matter of fact, it does not do so. Treatment based on Politzer's assumption did no good, *e. g.*, inflation, the pressure probe and vibratory massage. Fibrolysin was also used on this theory, but without any result. A more reasonable view is that according to which the extraneous noises are necessary to stimulate the atrophic nerve apparatus.

Jenkins and others hold that paracousis is more apparent than real; that it is due to the fact that extraneous noises like those in a train or street cars, which are low in pitch, do not disturb an otosclerotic so much as a normal-hearing person, and that therefore the otosclerotic gets more advantage from the raising of his friend's voice, which always occurs under such conditions. Jenkins proved this by getting someone to read to a normal person and an otosclerotic in a quiet room in which a noise was suddenly produced. The normal person did not notice that the reader's voice had been raised at all when the noise occurred, but the otosclerotic found it very marked.

Diagnosis. Can we be sure that a given case is one of otosclerosis? Certainly, if we have a family history of deafness, normal

drumheads, flamingo tinge of the promontory, Bezold symptom triad, normal Eustachian tubes and no improvement on inflation.

I have shown you, however, these cases in which otosclerosis was combined with middle-ear suppuration or with an adhesive process. How can we then diagnose otosclerosis? It is not so easy, but if we have marked or almost complete deafness, severe tinnitus, great elevation of the lower tone limit and paracousis it is at least extremely probable that secondary otosclerosis is present.

Jenkins points out that in middle-ear deafness, apart from otosclerosis, Rinné's test only becomes negative when the hearing distance is considerably reduced, *e. g.*, twelve feet for the conversation voice. In otosclerosis, on the other hand Rinné's test is negative even though the patient can hear the conversation voice at six feet. He also calls attention to the confident affirmative answer of the otosclerotic when asked if she can hear the fork on the mastoid after she has ceased to hear it by air conduction. This contrasts with the hesitating answer of the patient who has only an adhesive process in the tympanum. The same remarks apply to the lateralization of the Schwabach test.

Treatment. In Edinburgh, I am sorry to say, since we have learned to diagnose otosclerosis, we have not been very enterprising in the treatment of non-suppurative deafness. I have vivid recollections of the weary hours I spent in my first few years of otology in passing the Eustachian catheter and bougie, in massaging the drumhead with Delstanche's apparatus, in injecting fibrolysin and other useless measures. Still, I felt that something should, if possible, be done for these cases, and when I visited Vienna, in 1912, I asked Dr. (now Prof.) Neumann to give me a course on the treatment of non-suppurative deafness by means of galvanism, *x*-rays, radium, vibratory massage, auditory reëducation, etc. Dr. Neumann looked at me and said, "No, Dr. Fraser, I will willingly give you a course on Ohrenschwindel, but I will not give you a course on Taschenschwindel." I felt rather snubbed and asked if it were really "as bad as that." I had no wish to become a charlatan.

I do not know if this accurately represents the present state of affairs, but, if so, are we going to take it "lying down?" We may be willing to explain to an old man of seventy or eighty years that deafness is frequently produced by changes which occur in the inner ear as the result of *anno domini*, *i. e.*, of arteriosclerosis. We may be prepared to tell a patient with congenital or acquired syphilitic deafness that little or nothing can be done, for in my experience arsphenamine and its substitutes have no effect: Mercurial inunctions and iodides are more hopeful. Are we, however, to be content with saying to a young girl or a young man that the case is one of chronic and usually progressive deafness for which, in the present state of our knowledge, very little can be done, and to suggest lip-reading if the patient is young enough to take it up and is the kind of person who "looks you in the face?" If not, we can advise an artificial aid to hearing and write a short and unsatisfactory account to the patient's doctor in regard to the treatment of tinnitus. I hope to hear this evening that others are more successful.

Gray points out that there is no routine treatment for otosclerosis—every case must be a study in itself. He admits that the results of treatment, so far as improvement goes, are generally unsatisfactory. In a large number, especially in the very early stages, the disease may be arrested or greatly retarded. In a few some improvement may be obtained. If the affection comes on early in life the outlook is extremely bad. Cases of hereditary otosclerosis are unfavorable; if tinnitus is marked, the case is likely to progress rapidly. If the pure form of the disease, unassociated with catarrh of the pharynx or middle ear, a moist climate has no particularly bad effect, but severe cold is unfavorable. Gray lays great stress upon diet, and states that meat, which permits of considerable toxin formation, has a bad effect. Alcohol, tobacco, strong coffee and tea are also unfavorable. The injurious effect of pregnancy and the puerperium have been rather overestimated. Dieting, laxatives, intestinal antiseptics, the elimination of septic foci and vaccine treatment all have a place. We can do something better than merely tell the patient

to learn lip-reading or to use an artificial aid to hearing. The value of the former has been considerably exaggerated. Sir Robert Woods injects a preparation of iodine into the tympanic cavity with good results.

About twelve years ago Charles Heath, of London, England, published a paper in which he described his method of teaching cases of deafness associated with paracousis—cases which he described as those with loose ear-drums or pocket-handkerchief deafness. Heath believed that the pathology of this affection consisted in relaxation of the drumhead from too frequent and forcible inflations of the middle ear, inflations which caused the membrane to lose its proper tension, the tendons of the tympanic muscle to become relaxed and the labyrinthine tension to be unduly lowered. You will remember that Heath's method consisted in painting the drumhead at frequent intervals with increasing strengths of blistering fluid. At the urgent request of a friend of my own, a medical man, suffering from otosclerosis secondary to otitis media, I tried the method, but without any beneficial result. Gray treated twelve cases according to Heath's method, but in not a single instance was there any improvement.

The method of auditory reëducation associated with the names of Zund-Burguet and Maurice has also given unsatisfactory results according to the reports of Gray and of Muecke. My own experience is limited, as I did not carry out this treatment myself. Several of my patients, however, were treated by the late Major Porter, but without benefit as far as my observation went.

Lately, I saw a patient who told me that she had been diagnosed ten years ago as a case of unilateral otosclerosis. She had been informed that she would gradually become deaf, first of all in the already affected ear and later in the sound one, with all probability. I examined the patient and confirmed the previous diagnosis. I found normal drumheads, lengthened Schwabach, Weber to right (bad ear) and loss of hearing for C. 32 on the affected side. The voice test, however, showed that the patient could repeat the forced whisper at twelve feet from the affected ear while the left ear was normal for the whisper and watch. I

told the patient that I had never known such slow deterioration of hearing in a case of otosclerosis. The patient then informed me that at intervals during the whole ten years she had been treated by vaccine therapy, the vaccines being made from swabs obtained from the nose and nasopharynx.

Radium. My experience of this line of treatment only amounts to two cases, neither improved. The first was a girl of twenty-two years, a typical case, with a history of slight deafness since shortly after puberty, normal drumheads, flamingo-tinge of promontory, etc. Inflation had been tried without benefit. Four applications of radium were given to the right ear, but, though the meatus was protected by tinfoil, the only result was a radium burn of the skin lining the passage. The hearing was not improved. In the other case the result was also unsatisfactory.

X-rays. As far as I know very little work has been done in Edinburgh on the subject. Dr. Hope Fowler tells me that, following the advice of two colleagues in Manchester, he has treated several cases by cross-fire radiations. Some have apparently been benefited while others are stationary. He admits that the period of observation has been too short to draw definite conclusions. Prof. Hugo Frey writes to me that he is just about to read a paper on the x-ray treatment of otosclerosis. He seems to be hopeful regarding the method. I should be glad to hear the opinion of those otologists who have treated cases or have had their patients treated along the line.

Various operations have been performed for the relief or cure of otosclerosis. I will not refer to earlier methods, of which I have no experience, but will pass on to the making of a window in the bony wall of the posterior semicircular canal (Bárány) or in the lateral or even the superior canal. I have carried out two of these operations at the Royal Infirmary, in each case operating only on the worst hearing ear. In the first case the perilymph space of the lateral canal was probably opened and labyrinthitis resulted. The patient recovered, but of course became quite deaf in the operated ear. The second case was more successful. The bony cap of the lateral canal was removed and a skin graft

applied. The operated ear is now the better hearing ear, but the improvement was not very great, and, though this patient has sent on to me several others suffering from otosclerosis, I have always refused to interfere. It may be that the dissecting microscope used by Prof. Holmgren, of Stockholm, will enable us to operate on the labyrinth with greater accuracy. One feels vaguely that there must be operations on the ear to correspond with the iridectomy and cataract extraction of the ophthalmologist.

I have had no success with lumbar puncture in the treatment of tinnitus, though Babinski states that 33 per cent of cases were relieved by this procedure.

Our experience in Edinburgh in regard to the destruction of the cochlea in cases of unbearable tinnitus has been very bad. One case, already published, was that of a rather feeble-minded girl, while the other (not my own case) was a lunatic. In both the operation was followed by meningitis and death.

Conclusions. We are all aware of the famous reproach to otology that ear diseases are divided into two classes: (1) Those that anyone can cure with a syringe, and (2) those that nobody can cure and that should be sent to an ear specialist. This witticism is, of course, unfair, but there is just enough truth in it to give it a sting. We know that we can deal more or less successfully with suppuration in the middle ear and its consequences. Why cannot more be done for non-suppurative deafness?

The following questions suggest themselves for solution:

1. Is otosclerosis a pathological entity?

2. Is it congenital? Is it ever present in the labyrinth capsule of the fetus or infant? A coöperative investigation of the inner ear in a large series of cases of fetal death from placenta previa or breech presentation would settle this point. Manasse has examined 200 fetuses and in 11 found symmetrical islands of cartilage in the labyrinth capsule in the site of election. Is this a preliminary to otosclerosis? I have heard Gray say that in his opinion one person in every 200 suffers from otosclerosis, but if Manasse's observations are accepted, we should expect a percentage of 5.5.

3. Is otosclerosis an inflammatory affection and does it follow otitis media? Can it be distinguished sharply from middle-ear catarrh?

4. Is otosclerosis a degenerative or wasting disease?

5. Is there any connection between disorders of the endocrine glands and otosclerosis?

6. What is the cause of deafness in otosclerosis, seeing that in early cases, at any rate, there is no bony ankylosis of the stapes?

7. What is the connection, if any, between otosclerosis, nerve deafness and congenital deaf-mutism?

8. How often is the vestibular apparatus involved in otosclerosis and why is there giddiness in one case and not in another?

If an investigation is organized I suggest that it should be divided into two parts:

(*a*) *Laboratory Work.*—Microscopical, chemical, experimental.

(*b*) *Clinical.* (1) Statistical: As regards age, sex, heredity, distribution, association with other diseases. (2) Symptoms: Clinical examination, diagnosis. (3) Treatment: Medicinal, ductless-gland therapy, vaccines, local non-operative procedures, operations.

The two parts of the investigation should, of course, be coördinated, *e. g.*, the clinical examination and postmortem microscopical examination.

Individuals have different gifts and have been especially trained along various lines. Some are clinicians, others laboratory workers; some of the latter have been trained as chemists, others as physicists or pathologists; some lean toward medicine, others to surgery; one man laboriously attacks a problem from the pathological and microscopical point of view, another has a brain wave and is carried to a brilliant and, let us hope, a correct conclusion. My suggestion is that in each country the problem should be tackled by a team composed of men who individually and together would represent all the different aspects.

A coöperative investigation can only be originated in America, which is the most universally popular—perhaps I should say the least unpopular—country in the world.

DISCUSSION

Dr. B. Alexander Randall: The chairman is looking pointedly to me to expose my ignorance on the subject, and I suppose as one of the elders I ought therefore to say a few words, if only in my warm admiration of what has been presented to us.

My experience of otosclerosis is small, which may earmark the inferiority of my diagnostic methods. I see a great many cases so diagnosed by others, but fail to recognize that such diagnosis is correct.

I regard, of course, the Bezold triad as belonging to this as to almost all of the chronic non-suppurative deafness cases, but cannot see that it has more point to it than that, or that it should be regarded as in any sense diagnostic. Naturally using that as a criterion, Bezold saw otosclerosis cases by the thousands or by the hundreds. I have seen about 30 cases in thirty years. Of course, I do regard the sign of a reddish promontory as a very marked and probable point in the cases, and where that is absent would very gravely question the correctness. If the case is in full bloom in other respects, and apparently advancing, I should feel that it was a pseudo-case of otosclerosis and not a true one.

The claim of Politzer and others that there is no involvement of the mucosa of the tympanic cavity has never struck me as probably correct. Not only do we have the combined cases so well shown us by Prof. Fraser, but I believe that in those cases which are as pure otosclerosis as we are apt to find, unless it be such as Alexander has recognized in the embryo kitten, which I regard as a huge joke. I regard the presence of mucous-membrane involvement as generally to be found if properly sought for. Of course, one may say that a normal mucosa covers the promontory which glows cherry red through the fairly normal drumhead, and the drumhead is frequently so normal that every detail of the tympanic cavity can be seen through it.

I question, however, how far we are clearly familiar with normal drumheads. I remember a fairly prominent man of this city who sent me a patient a few years ago and asked me what in the world was the matter in that girl's case, that there was a sheen of something like cotton in the meatus, and he could not make out what the trouble was. I told him she had a normal drumhead, and he had probably not seen one before. I think those which we accept as decently normal drumheads are often very far from being strictly so.

The point that has always seemed to me of distinct interest as a diagnostic sign, and as bearing upon this particular question, is that little sign put forward by Lowenberg, many years ago, that, if we inflate chloroform vapor into these ears, red though the promontory is, as we see it,

instead of the hot, burning sensation common to most normal ears, we get a distinct sense of coolness, almost cold, from the chloroform vapor, but we are told this is a strictly normal mucous membrane to which chloroform vapor feels cold.

As to the amount of treatment, I have not been able, in the few cases that I have seen, to take the extremely pessimistic view which has been set forth by many men or to accept with more than a very large grain of salt the beautiful family trees which have been presented, most of them almost exclusively figments of the imagination of the writer who saw none of the older members of the family and had no positive means of diagnosticating the genuineness of the otosclerosis that he did see.

That a woman with a family history bad, and with signs of otosclerosis or suggestions of it, should be absolutely held back from marriage as being injudicious for herself as almost sure to provoke an advance of the trouble, and almost sure to pass on this disease to her children, I think is a very distinctly overdrawn matter; yet it has been put forward with great vigor by some men who have written and talked on this subject. Certainly in the experience of these 30 odd cases which I have seen there has frequently been some decided improvement under treatment, and there has sometimes been fairly maintained improvement in spite of marriage. I cannot feel that I would do more than warn such patients that there is a strong feeling of that sort among other men, and let it go at that. I cannot say that I would urge it as being my own view. That these cases have been frequently apparently much helped by treatment and that treatment fairly vigorous has been experienced probably because I was dealing with the mixed cases and was helping the catarrhal element in the matter—and perhaps harming—but I could not see that I harmed the ossicles. I cannot see myself as a pessimist in the matter, although I feel that I am a chronic optimist, and that I am open to criticism as such. That these cases are very learnedly talked about by my youngers has not convinced me that my skepticism as to many points is altogether in the wrong, or that my optimism is one that is likely to do more harm than good.

Dr. S. MacCuen Smith: First, I would like to say a word about these most excellent slides: It is probably news to some of my hearers at least, that Dr. Fraser was good enough to bring a complete set of slides, including these that he has shown this evening and others to this country, and they are to be presented to one of our American cities. I do not know just how that is to be decided or whether it has been decided. I should be very glad, indeed, if Dr. Fraser would let us know. The point seems to be general, as far as I am concerned individually, that if I would have those slides I would almost necessarily have to have an interpreter to

point out just what the slides represented in a good many instances. That is owing to the fact that we have had in this country very few men who have had any experience in this line. The only one with whom I am acquainted who might probably decipher these normal slides is Shambaugh, of Chicago. The one thought was that they should go to Chicago; the other one was that they should go to New York on account of the large amount of money that has been given to the Post-Graduate School there. Naturally, those of us in Philadelphia, having in mind the College of Physicians, thought that we should have them here. If we have no one in Philadelphia to interpret them, would it not be a good thing to have one of our younger men schooled in this particular line of work?

One question I would like to ask Dr. Fraser in regard to what might be determined otosclerosis in pregnancy—and the reason that I ask it is to find out whether the case I am about to describe was a case of otosclerosis.

It so happened that I knew a young woman who was married, and with her first child, she became very deaf. Having in mind some other cases, and she being in a position to do anything possible to prevent deafness, during her second pregnancy she went to a mountainous region and stayed there until it was just about time to give birth to her child, and then returned to Philadelphia for that purpose. Some little time after the child was born she regained her hearing. This same thing happened in 4 other pregnancies with the same result. In the last pregnancy, however, recently, she did not go away because she did not have any impairment of hearing. Whether this was due to some pathological condition other than otosclerosis I should be very glad to have Dr. Fraser enlighten me.

It is rather interesting to hear Dr. Fraser speak of the various types of otosclerosis. Of course, we recognize the primary and the secondary, but personally I feel that in order to speak of a case of otosclerosis we have to have the normal drumhead and practically the tympanic cavity and tube, etc., and yet I have come in contact with quite a few of the cases between the ages which Dr. Fraser mentioned and which seemed to be otosclerosis.

Regarding the hereditary causes, which brings in the family tree which Dr. Randall has just spoken about, there must be something in heredity. I happen to know a family, whose name was Wilson, in which every member for probably five generations who represented the mother's side of the family, and who during these five generations spoke like the Wilson side or looked like them, had at an early age become deaf. I happened to know this family very well, because later on they became related to me by marriage. I took three or four of those children, before

they arrived at this age, and sent them to Switzerland, and they lived there and were educated and looked after by a man I knew in Switzerland. Every one of these children developed deafness and tinnitus aurium and the symptoms of otosclerosis after the age of about eighteen. If Dr. Fraser could give us some little thought on that I should be very much obliged to him.

As to the experimental investigation, I believe that is the only way. It is international, and the very best way that we can probably find out something about the pathology of otosclerosis, and if we can get something more about the pathology, it is probably the first thing we will have to determine before we can hope to be of any service. I might say in this connection that at the last meeting of the American Otological Society, which has just recently convened at Atlantic City, ten dollars was authorized as an assessment from each member of the society as a fund to start the investigating of otosclerosis, the thought being that perhaps the American Laryngological, the American Triological and the American Academy of Medicine would all follow in line. The underlying thought of this all was not that the money which would be given by members of our specialty in various parts of the country would be sufficient to be of any service, but that if we showed a certain amount of interest and were willing to give some money, individually and as a society, we probably could interest the Rockefeller Foundation; and two or three of the men—more particularly Dr. Dench—thought that this was the better way to do it, and he thought that the Rockefeller Foundation would take it up. It was thought at that time that we would at least have to pay a man for this investigation what a professor of pathology would be likely to receive in an institution.

Now as to treatment, personally I am not able to help these cases very much, and that brings up a very interesting question on which I would be very glad to have an expression of opinion from Dr. Fraser. Even though we cannot help these cases, are we justified in taking a case, either boy or girl, or man or woman, about eighteen to twenty-five or thirty—are we justified in saying to these people who are suffering not only from a given impairment of hearing, but especially from tinnitus aurium (the vast majority of whom, if they could, would only get rid of the noises) are we justified under these cirumstances in telling these people that they cannot be helped? I believe, as Dr. Randall has said, that a certain number of these cases can be benefited somewhat. I think the tinnitus aurium can be helped. Are we justified, therefore, in treating these cases and not telling them promptly that we cannot do anything for them?

Dr. Francis R. Packard: I would like to ask Dr. Fraser if there is anything in the line of treatment that has come to his attention that has been particularly harmful in these cases. It seems some people say that mixed treatment is worse than no treatment, and there seems to be an impression among the patients that they have been harmed. I would like to know whether Dr. Fraser thinks that any distinct harm has been done, for instance, by massage or inflation of the Eustachian tube, or anything else, which influenced the otosclerotic process in a true case of otosclerosis?

Dr. E. B. Gleason: The paper is so complete that there is very little to be said in the discussion. Dr. Randall referred to inflation of the middle ear with chloroform in these cases of otosclerosis. Occasionally we find that one ear will feel warm after the inflation by the chloroform method with chloroform vapor while the other will get cold. After you have done your utmost in the way of diagnosis, I think the prognosis is very much aided by what occurs in the patient. If after a routine treatment, consisting of inflation with the catheter or by the Politzer method or by massage, the patient, instead of hearing better, has his hearing not improved at all, and still more if his hearing is made distinctly worse, that patient will not probably be greatly improved by local treatment. On the other hand, for the time being, you will get a certain amount of relief from the tinnitus aurium by the administration of chloroform. I am distinctly of the opinion that very distinct harm, possibly as the result of the increasing congestion, is done by a continued course of treatment of the middle ear in these sclerotic cases, consisting of inflation and massage.

Dr. Smith: Might I add one word to what Dr. Gleason has said, carrying out the thought that if the patient is benefited by the inflation, that this is a case in which the patient is benefited—I quite agree with that; but I wonder whether, after all, we do not feel that these cases of otosclerosis are atrophic, and we would hardly expect to have any improvement by inflation. If they are hypertrophic, those cases would rather naturally be benefited by the inflation and treatment.

Dr. Randall: I would like to ask Dr. Fraser to inform us if, in his findings, he has found the cases which feel worse, as Dr. Gleason has suggested, are not almost always pretty clear in their evidence of nerve deafness.

Dr. Nathan P. Stauffer: I would like to ask Dr. Fraser his explanation of the noises.

In regard to heredity, I have had five in one family; all have been deaf and all on the father's side. All attributed it to heredity traced from the grandfather down.

I would also like to ask Dr. Fraser if he has noticed any pain in these patients. Why should a nerve die without having some pain? We have it in our teeth and other parts of the body.

Dr. Smith: Might I have just a moment to repeat something that I have said on former occasions to this effect, that formerly it has been our custom, especially men of my age, to go to Germany for our instruction. Dr. Fraser has been good enough to have made the statement on one or two occasions during his visit here to the effect that he or some other members of the English-speaking race thoughout the world would feel probably that they could come to this country and receive some instruction or some benefit in a medical way, and I just want to make it appear that a closer coöperation between the English-speaking people would be of great benefit not only to us but to the world. In other words, those of us, I think, who have heard Dr. Fraser, would feel that we wanted to learn something a little more intimately in this line of work, and that is true also of the nose and throat work which is being developed on the other side by Logan Turner and others, so that we would very naturally feel like going to Great Britain and getting some instruction, not with a view to leaving out the German nation, because in some instances we would probably all go to the German nation, but I believe it would bring about a better coöperation between the English-speaking people.

Dr. George Fetterolf: The Mütter Museum has a fund for acquiring just such specimens as we have seen here tonight. I shall bring before the Mütter Museum a plan for securing a specimen of every one of Dr. Fraser's slides that can be obtained, and if those of you who would like to see such slides will keep in touch with the Museum, just as soon as they are purchasable, the matter will be brought up before the Committee, and we hope that these plans will be realized.

Dr. Fraser: In reply to the discussion, first of all with regard to what Dr. Randall said: Dr. Randall laid stress on the difficulty in diagnosis, and I admit that there are many cases which I regard as otosclerosis, and Dr. Logan Turner does not think that the otosclerosis or tinnitus is proven but there are many cases besides otosclerosis to which the same thing applies. Dr. Randall doubts the value of the Bezold symptom triad. Well, it is quite true that in cases of obvious middle-ear trouble you do

have a negative Rinné. You do have lengthening of bone conduction, and perhaps you have lost say C 16 or 32, but certainly if one has with that Bezold symptom triad in the presence of a normal tympanic membrane, then I say otosclerosis is certainly present. Even if the tympanic membrane is not normal, I think it is a matter of degree. If the patient can hear 32, and even 64 or 128, if the Rinné is markedly negative, as Jenkins points out, when your patient has ceased to hear one of the middle forks, say C 56, and then you put it on the mastoid and the patient's face lights up and he says, "O yes, I hear that," then with a marked raising of the lower tone limit, I think you can be certain if there is anything like a family history of deafness you can be tolerably certain that otosclerosis is present.

I point out to you that out of 7 cases examined, 4 by Dr. Gray and 3 by myself, 5 of the cases had middle-ear trouble, so that you cannot say that otosclerosis is a disease in which there is a flamingo tinge of the promontory. Then if 5 of these 7 cases examined had otitis media, if that is anything like the proportion, we know that we have quite a number of cases of otitis media where the drum membrane is not normal, and then you would arrive at a very considerable number of cases existing in the world which had otosclerosis.

I am sorry to state my ignorance that I did not know about this chloroform test being tried, and I cannot say anything about it.

With regard to marriage, I certainly agree with Dr. Randall. I do not think that we know enough to forbid a girl with otosclerosis from marrying. The position I take is that if the girl coming from an otosclerotic family is going to marry, she certainly should marry into a family with perfect hearing and not into a family with deafness. Then, according to the Mendelian laws, we would think that her children if they kept out of a family with otosclerosis would not have otosclerosis to any great degree.

I am glad to hear that Dr. Randall has seen improvement under treatment. I rather confess that I have not found success.

You remember Mr. Charles Heath, of London, introduced a method of painting the tympanic membrane with Listerine fluid mixed with tincture of lavender. This gave a nice, reddish tint to the mixture, and he painted the membrana tympani first with weak Listerine fluid and then increased the strength. The meatus cast off epithelium, and there was some discharge. This was done almost every day and went on for some weeks, and then the treatment was stopped and the patient began to hear better. Of course, the patient had been made considerably worse, and when the treatment was stopped the hearing improved. I did carry out this treatment once at the urgent request of a doctor who suffered from otosclerosis,

and Dr. Gray found the same result. We have had the other lines of treatment, as the bougie, etc. Then, again, Dr. Mukie, one of the otologists at the London Hospital, tried out this method and found it was not good at all. Fibrolysin I have injected and combined it with massage and vibration and could not get any improvement at all. I have also injected, according to Mr. Lake's method, some kind of mercurial preparation with no result. So I have been very pessimistic in regard to treatment. I have only been saying this to the patient: At present we really do not know anything that will help. There are hundreds and thousands of cases like yours in the world. When anything is discovered you may be quite sure that it will appear in the daily mail and all the papers.

Dr. MacCuen Smith has kindly spoken about the microscopical slides and the lantern slides I was going to present to some society in this country. I might say that these include the anatomy, so that there should not be any difficulty in many familiarizing themselves with the slides. I will be extremely delighted if you could get a few disciples; and if anybody would like to know the method of carrying out this way of examining the inner ear I would be only too glad to show anybody or send a printed account of the method. I might arrange to do it sometime here. All you want is a temporal bone or two, a saw and a pair of bone pliers and a scalpel, and I could easily show you how to carry out the method. Dr. Haskin, the secretary of the Triological Society, is making arrangements with somebody who makes lantern slides to have a set of specimens reproduced and, as it were, put on the market. The operator said that if a sufficient number were ordered the slides would only cost about twenty-five cents each. If there were only a few sets ordered they would be about thirty-five cents each. I think about one hundred slides would give you the whole anatomy and pathology of the inner ear as far as I have been able to make out. It ought to be quite easy to multiply these sets, so that anyone who wanted them could get them.

I am very glad, indeed, to hear that there is going to be a fund raised to establish a research worker, and I hope that the pathological aspect is not to be the only one. It will take six months before the man gets going with his pathology. It takes me six months from the time the patient dies to get this. There is a fixation about three weeks after death. The calcification takes six or eight weeks. There is the washing in water, the imbedding in thin saline for a month, then leaving the lid off the bottle, etc. The whole thing takes six months, so even if the research worker were lucky enough, it would be six months before he would be able to get his first section cut and stained. So I hope he would be somebody who would be prepared not only to work along the pathological line but also the clinical or statistical or some other method.

With regard to what I say to the cases, I think I have already answered that. The tinnitus is certainly a difficulty. I was talking this evening over this matter and someone was telling the story of the bug. It is a story of somebody who had been traveling in a train in this country and had been bitten, and had written a very angry letter to the railroad company and received a most beautiful typewritten letter in reply, explaining how steps were going to be taken to have the carriage put right. The man was delighted with the answer, but in turning over the sheet he found pinned to the back of it his own letter and a note in pencil, "Send this boob the bug letter."

Gentlemen, I also have a "bug letter" that I use when a doctor sends me a case of otosclerosis, explaining the tinnitus. I have to write him a letter stating the bad nature of this business, and I get so tired of dictating this letter to my secretary that I said to her one day, "We also will have a 'bug letter,' so that whenever a doctor writes regarding otosclerosis we can copy that out and I will be saved the trouble of dictating it in the future." It is a very difficult matter. I remember once treating the sister of a doctor from America. She was working at a school in Edinburgh. I told her plainly and candidly, as I usually do, about the disease, because she was going to be a teacher. I do not think it is suitable for a girl who is probably going to get more deaf that she should take up teaching. I do not think teaching or stenography are suitable for anybody who has this increasing deafness, and I received a most angry letter from the brother in America, who told me that I should be ashamed of myself for telling her what I had. I think it is better to tell patients so that they may be guided and plan their lives accordingly.

With regard to operations for these cases, I certainly would not do any operation on the nose or throat if the membrana tympani is normal and the tubes open. If the tympanic membrane is normal it cannot possibly do any good to operate on the nose, however crooked the septum may be; and I think it is not right to operate on the septum and tell the patient that it is to prevent the deafness going to the other ear if only one ear is affected.

With regard to the focus of infection, my own experience is that I could not find much in focal infection. Most of these sad cases are in young girls of from eighteen to twenty-six. I look at their teeth with the naked eye, and can see nothing apparently wrong. I can see nothing in the pharynx to account for it. But, of course, there are many other parts of the body where they may have a focus of infection.

I quite agree that if the patient were made worse by inflation it would be foolish to go on.

I believe that Luetche was right in that you cannot make a diagnosis between chronic middle-ear catarrh and otosclerosis.

With regard to the causes of tinnitus—that is difficult, but I think the best explanation is that the patient is hearing the noises of his own circulation. Under normal conditions the labyrinthine capsule is surrounded by this hard cartilage and bone. It is, as it were, sound-proof, and if in the walls of this sound-proof box this area of spongy bone appears, with the dilated bloodvessels, I think that is very probably the cause of the tinnitus.

I have had cases with pain, but only occasionally. I do not know that I have made up my mind as to how important these nerve changes are in otosclerosis, but I do not think that pain is a symptom coming from the eighth nerve. If the eighth nerve is affected, tinnitus and giddiness in the eighth nerve correspond to pain in the fifth nerve.

OCTOBER 17

Tonsillar Pathology in Relation to Systemic Infection

BY GEORGE B. WOOD, M.D.

The size of the faucial tonsil and the fact that it is so frequently the seat of pathological conditions demanding its removal, have made it the chief lymphoid deposit of the throat for study and investigation. However we must remember that all the lymphoid tissue of the pharynx and of the gastrointestinal tract is similar in structure and probably possesses similar physiological properties, and, perhaps, to a lesser degree, similar morbid tendencies.

The faucial tonsil is separated from neighboring structures by a fibrous capsule, from which trabeculæ pass into the parenchyma of the tonsil, carrying the bloodvessels, nerves and efferent lymphatics. There are no afferent lymphatics. Between the trabeculæ is the parenchyma proper of the tonsil, consisting of the germinating follicles and the interfollicular tissue. Into the parenchyma is projected, from the free surface of the tonsil, ingrowths of epithelium which, being hollowed out by a desquamation of the central cells, form the crypts. The epithelium covering the surface of the tonsil is of the squamous, stratified type, with a distinct

basement membrane, and beneath this is a definite layer of fibrous tissue. As soon as the epithelium dips inward to form the crypt the subepithelial fibrous layer disappears, and also the greater part of the basement membrane, so that the basal cells of the epithelium become intimately associated with the interfollicular tissue. The cryptal epithelium is so infiltrated with lymphoid cells that usually there are only one or two layers of intact epithelial cells separating the cryptal lumen from the parenchyma.

The histology of the tonsil presents many very interesting cytological phenomena which will reward the student or investigator for any amount of detailed study. It is not my purpose, however, at this time to delve deeply into this side of the question, though I want to briefly discuss certain structures which are often supposed to be of pathological significance, but are found so frequently in the tonsil that considerable debate has arisen as to their true import.

The plasma cell is found in varying numbers in practically all tonsils in individuals over two or three weeks of age. David J. Davis says that they are not found in the fetus or newborn and are more numerous in hypertrophied tonsils than in supposedly normal ones. He believes that their presence probably indicates a chronic infection of the tonsillar crypts. Goodale found them abundantly in acute tonsillitis, and, oddly enough, more numerous in the parenchymatous type of the disease than in the suppurative. J. Gordon Wilson has made a very careful study of these cells and is not at all certain that a bacterial irritation is necessary for their appearance. He feels rather that they are the manifestation of some metabolic activity and that they have an important function to perform, namely, to combat certain toxic bodies, which bodies may be of katabolic origin as well as bacterial. However, it would seem probable that their presence in unusual numbers is an evidence of an unusual toxic attack, most plausibly of bacterial origin.

Eosinophile cells are occasionally found in tonsils just beneath the epithelium. According to my own observations, they are apt to be very abundant in acute inflammations of the tonsil, but

the significance of their presence is not known. The presence of a few scattered eosinophile cells I do not believe could be interpreted as an indication of a morbid condition.

In practically every tonsil that is examined histologically we will find polymorphonuclear cells. Their presence in any great numbers or in concentration, must be accepted as an indication of some toxic irritation due to bacteria, but if we would call every tonsil diseased in which they are found, we must then call every tonsil pathologic.

The amount of fibrous tissue in a tonsil varies considerably according to the age and also individually. While a marked fibrosis must be considered as evidence of previous inflammation, it is very difficult to know just where one should draw the line between a physiological fibrosis and a pathological one. It must be left to the opinion of the examining pathologist. We should remember, however, that fibrosis is a scar of past trouble and not an indication of present active disease.

Cartilage and bone are frequently found in the capsule and trabeculæ, but there is considerable dispute as to their pathological significance. A number of observers, among whom are J. Forman, H. Albert and M. G. Douglas, believe that the cartilage and bone are the result of a metaplasia, and as such are evidence of a chronic inflammation. On the other hand, H. Mantchik asserts that the cartilage is the result of a defective retrogression of the primitive cartilaginous skeleton of the tonsillar region. In this view he is supported by Grosvenor, who says that the condition is not one of metaplastic change but is probably a development of the matrix of unused or displaced embryonic cells derived from the second branchial cleft. E. G. Carey also believes that the cartilage is an embryonic derivative. Imhofer says that this cartilage results from both causes, *i. e.*, it may be embryologic or it may be due to an inflammatory metaplasia. In a series of 696 tonsils reported by four different observers, these cartilaginous depositis were found 87 times. They have been found during embryonic life and may occur in tonsils which are otherwise free from inflammatory evidence. The structure is usually that of

embryonic cartilage. The bony tissue which is frequently found in association with this cartilage is most plausibly the result of a physiological process of ossification.

The life-cycle of the tonsil does not correspond with the life-cycle of the individual. The tonsil reaches its full tide of functional activity at a very early age and then tends to undergo a type of involution. At the time of the highest functional activity there is no doubt that the cryptal epithelium is in a state of growth. The basal cells are apparently penetrating deeper and deeper into the interfollicular substance and the surface cells desquamating. This desquamation of the epithelium gives rise to a type of lacunar plug which, at least in the deep terminal portions of the crypts, must be considered a normal formation, but with possibilities of pathology which we will discuss when we consider positive diseased conditions.

There have been several attempts to classify by name the chronic pathological conditions of the tonsil, some of them based on the clinical appearances only and others on the pathological examination. Except for a few well-recognized conditions, such as tuberculosis and hyperkeratosis, it is exceedingly difficult to do this because of the variety of lesions that may be found in one tonsil. The classification, originally suggested by Semon and Williams, and quoted by J. J. Moore, has been followed by a number of men, but it is not entirely satisfactory. In this there is distinguished first a chronic cryptic or lacunar tonsillitis; second, a chronic interstitial tonsillitis; and third, a chronic peritonsillitis.

Under chronic lacunar tonsillitis are placed all tonsils that show changes of the lining, size and contents of the crypts such as irregular outline of the epithelium, desquamation and keratinization of the superficial cells, dilatation of the crypts from the accumulation of desquamated epithelium and the presence in the crypts of cholesterin crystals, polymorphonuclear leukocytes, hyaline material and bacteria.

The term chronic interstitial tonsillitis includes all of the tonsils which show a pathological increase in connective tissue or the presence of cartilage and bone in the connective-tissue stroma,

and the author has for some reason associated with this group the hypertrophic tonsil.

Under chronic peritonsillitis are those tonsils which show an increase in the thickness of the capsule or a deposit of fibrous tissue in the peritonsillar region as a result of a previous peritonsillar infection.

While such a terminology is of some value for the purposes of classification, it helps little in defining the condition which had been determined by the pathological examination and of rating its possible influence on the general health. The chronic interstitial tonsillitis and the chronic peritonsillitis do not represent active pathological conditions, as they are simply scars of a previous inflammation. Because the hypertrophied tonsil was supposed to later on develop an excessive amount of fibrous tissue, they have been placed in the chronic interstitial group. I do not feel that this point is well taken, because even if we admit that a hypertrophic tonsil is a diseased condition, it is the parenchyma that is involved, and further, I have not seen any evidence to indicate that a hypertrophied tonsil will develop a greater percentage of fibrous tissue than a small tonsil. I might say here that to call a tonsil hypertrophied has to my mind little significance, except to indicate that the tonsil examined is larger than the average. Under the term of chronic lacunar tonsillitis there are included certain conditions which are undoubtedly physiological and normal, and others the presence of which is of no pathological significance.

From our present knowledge of the pathology of tonsillar infection, it would appear impossible to present a definite terminology which would adequately describe the pathological findings in a given tonsil. It would be far better if a pathologist in reporting on a tonsil condition were simply to note the conditions found without attempting classification. Happily this is what the majority of our pathologists are doing today.

In considering the various lesions which may be found in the tonsillar structures, it is evident that they must be placed in two groups:

In one group, which might be called the cryptal, the pathology is peculiar to the tonsil itself, because the anatomy of the tonsil makes possible certain conditions that can be found only where this type of anatomy exists. The crypt of the tonsil, with deep penetrations, with a peculiarly disintegrated epithelium, makes for the production of pathological conditions which, while really external to the body structure, produce an influence on both regional and distal body tissues.

In the other group, which might be called the parenchymatous, the lesions are beneath the cryptal epithelium and affect the tissues locally and distally in the same way and manner as similar lesions would in other portions of the body. There is this difference, however: The tonsil parenchyma is more easily reached by outside influence than any other subdermal structure, and this because the epithelium of the crypt does not offer as great a mechanical barrier to the penetration of toxin and its progenitor, the bacterium, as does other surface epithelium.

The contents of the tonsillar crypts vary exceedingly both in amount and character, and this variation is applicable not only to the different crypts but also for the individual crypt at different times. While the debris found often possesses pathological potentiality, yet the keratinized cells which frequently fill the lumen of the deep terminal portions of the crypts must be regarded as a normal condition. Extensive accumulation of desquamated cells is often sufficient to cause quite a marked dilatation of the crypt. These masses closely resemble cholesteatoma and the picture is made more perfect by the finding of cholesterin crystals. This condition in a sense is pathological, but it has no influence on the health of the tonsil or of the individual until it becomes infected by bacteria from the mouth. An evidence of such infection is the presence of polymorphonuclear cells. If the contents of the crypt show large numbers of polymorphonuclear cells, and they can be seen passing through the epithelium, then I would assume that the bacteria of the crypt are elaborating toxic material which is being absorbed through the epithelium. On the other hand the mere presence in the crypt of various forms

of bacteria cannot be looked upon as a pathological condition, as they are frequently found with no evidence of reaction in the tonsil. I further believe that large plugs of desquamated epithelium may undergo putrefaction in the tonsil crypt without inducing pathology, the infecting organism being saprophytic instead of pathogenic or of a pathogenesis to which that particular tonsil is immune.

Bacteria in the lumen of the tonsillar crypt can produce lesions of the tonsil, and consequently systemic reactions, either by their toxins being absorbed through the cryptal walls or by the bacteria themselves gaining entrance to the tonsil parenchyma. For instance, the early enlargement of the tonsil in acute tonsillitis is due to cell proliferation, which cell proliferation is the result of tissue reaction to the toxins absorbed from the crypt. Later on, after necrosis of the cryptal epithelium has taken place, the bacteria gain entrance and cause suppuration, which, as has been shown by Goodale, invariably starts in the germinating follicles. It is possible that, under proper conditions, certain of the pathogenic bacteria may pass in from the crypts through the normal epithelium. This has been proven by experimental research, but a careful observance of a large number of tonils had led me to feel that in the great majority of cases the bacteria will not pass beyond the first layer of epithelial cells as long as these cells retain their vitality.

It has been reported by several observers that the cryptal epithelium often shows complete breaks in its continuity, but I believe that the majority of the so-called breaks are artefacts, either produced during the removal of the tonsil or during the hardening, embedding and cutting of the specimen. On the other hand, small localized areas of acute inflammation, unrecognized clinically, are frequently found in the course of the regular routine tonsil study. In these small localized areas we have a counterpart of conditions found in acute tonsillitis. The crypt is filled with epithelial debris, polymorphonuclear leukocytes, fibrin and bacteria, and there is, perhaps, necrosis of the epithelial cells. When this latter takes place, we do have an open gateway for the bacteria to enter the tonsillar structures.

The accumulation of debris in the tonsillar crypts, the foundation of the debris being desquamated epithelial cells, is due either to poor drainage of the crypt or to an excessive growth of the epithelium. It is easy to understand how the anatomical variation of the individual crypt, or the shape of the tonsil as a whole, may interfere with the drainage. It is not so easy to explain why the epithelium at times tends to desquamate and at other times is entirely free from such tendency, but that such variation of growth of the epithelium does happen there is no doubt. For instance, in acute tonsillitis we notice an increase in the penetration of the basal cells into the interfollicular tissue, and we can find under these cirumstances mitotic figures showing strong evidence of growth activity. Again, in the condition called hyperkeratosis, a portion of the cryptal epithelium gives rise to the formation of cornified cells, the accumulation of which produces a horny projection from the crypt mouth, which is the pathological peculiarity of this condition. It would not seem unreasonable to believe that under certain circumstances a lacunar plug is the result of some stimulation that has been imparted to the epithelium of that crypt by bacterial toxins. Arguing from this standpoint, one would be justified in regarding any great cryptal accumulation as pathological. However, I believe that unless polynuclear cells are present, either in the surrounding epithelium or in the lumen of the crypt, the lacunar plug is not a menace to the health of the individual.

Sometimes the lumen of the crypt external to the accumulation becomes absolutely occluded, giving rise to a so-called retention cyst. If this cyst contains living pathogenic organisms, there is, of course, more chance for absorption of toxic products than if the crypt were open. In fact, we would expect, and it often does happen, that an acutely infected occluded crypt produces so much reaction that we have an extension of the suppurative process with definite abscess formation. On the other hand, these occlusion cysts often show little evidence of any inflammatory reaction, and if they have existed long enough to be placed in the category of chronic lesions are apt to be sterile, the bacteria prob-

ably having succumbed to the phagocytic action of the polymorphonuclear leukocytes.

An interesting anatomical freak is the occasional occurrence of mucous glands in the capsule of the tonsil, the ducts of which glands open into the deep end of one of the tonsillar crypts. The secretion from these glands would, of course, have a direct influence on the cryptal contents, though the condition cannot be classed as a pathological one, as I have noted certain writers do.

There are very few chronic pathological entities of the true parenchyma of the tonsils that can be recognized. Primary tuberculosis has been found to exist in about 5 per cent of all tonsils that were examined after operation. The importance of these lesions to the general health has been variously estimated by different observers. They are, of course, no more apt to lead to a general tuberculosis or a pulmonary involvement than are tuberculous glands of the neck.

Occasionally follicular abscesses are found in what are supposed to be chronic tonsillar conditions. D. J. Davis has four times seen small abscesses deep in the tonsillar tissue, near the capsule and surrounded by a dense fibrous wall. Hambrecht and Nuzum state that minute subepithelial abscesses are quite common, but I have never seen them. The reason that we do not see more evidence of chronic inflammation in the parenchyma of the tonsil is probably, in part at least, due to the lymph current that undoubtedly exists in the interfollicular tissues, preventing the stagnation of any toxic material. Toxins which have entered the parenchyma from the crypts are either absorbed by the venous capillaries or else removed by way of the efferent lymphatics. It is very important that we realize the possibility of soluble toxins being absorbed from the tonsil directly into the blood stream. If this takes place, and I am convinced it does, there can be a systemic effect without a preliminary involvement of the lymph nodes. The very great general intoxication which occurs so early in an attack of tonsillitis can only be explained by this assumption. Bacteria, on the other hand, are probably removed entirely by way of the lymph vessels, though a few very unusual organisms,

such as the anthrax bacilli, do penetrate the bloodvessel walls. The fact that they are seldom found in the interfollicular tissues and that the abscesses found in acute tonsillitis always originate in the follicles is evidence that bacteria do not accumulate or stagnate in the interfollicular tissues, at least for a sufficient length of time to produce necrotic changes. In my studies with the anthrax bacillus the organisms were found surrounding the follicles and filling the efferent lymphatics of the trabecula, but seldom in the interfollicular tissue.

It is very probable that a certain type of enlargement of the tonsil is due to proliferation of lymphoid elements, which proliferation may be the result of reaction to toxins absorbed from the crypt. How far this proliferation is responsible for chronically enlarged tonsils is problematical. While there is no doubt that some tonsils are normally larger than others, a distinct depreciation in the size of the tonsil can frequently be brought about by treatment directed toward the crypts. The shrinkage following such treatment, however, is always limited.

The frequency of occurrence of distinct pathological lesions in the tonsil is a most difficult question to answer with accuracy. Hambreacht and Nuzum made a careful study of the tonsils from 218 patients and claimed to have found definite pathological changes in 93 per cent. They used the classification of Moore and found chronic lacunar tonsillitis in 42 per cent; chronic interstitial tonsillitis in 21 per cent; chronic peritonsillitis in 6 per cent; abscesses (their location not being mentioned) in 10 per cent; lymphatic hyperplasia in 14 per cent; mucous glands in the capsule, 7 per cent; and cartilage in the capsule, 3 per cent. It is evident that a large number of these cases were not pathological in the sense that the condition found was a menace to the individual, such as mucous glands and cartilage in the capsule or of a hyperplasia of the connective tissue. Further, we must remember that the tonsils so examined were from individuals who had supposedly presented clinical symptoms sufficient to warrant tonsillectomy.

In closing, I would like to call attention to a peculiarity of the tonsil that has frequently impressed itself upon me, and this is

its tendency to rapid repair and its comparatively high degree of resistance against morbid processes. In experimental work with tuberculosis I noticed that the disease progressed much more rapidly in the lymph nodes of the neck than it did in the tonsillar structures. Also, a characteristic feature of primary tuberculosis of the tonsil is the absence of caseation. In acute tonsillitis the rapidity with which a tonsil riddled with suppurative foci becomes normal in size and color when immunity has once been established is rather remarkable. That it is able to withstand the almost constant insults that are heaped upon it by the flora of the crypts is further evidence of its disease-resisting ability.

Yet we must remember that because of the deep cryptal pockets and the peculiarity of its epithelium the tonsil does present, under some circumstances, a comparatively open gateway for infection to reach the interior of the body.

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The Bronchoscopic Treatment of Pulmonary Suppurations and Bronchial Asthma

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AND

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(By Invitation)

Etiology. At this time not a little discussion has been aroused as to the causative factor or factors in pulmonary abscess. No dissension from the three modes of infection is found, namely, (1) lymphatic extension, (2) blood-stream infection or (3) aspiration.

Lymphatic extension is generally considered a rare mode of pulmonary infection, but blood-stream infection and aspiration are not so easily disposed of.

The late outstanding articles bearing on this subject are those of Fetterolf and Fox,[1] Cutler and Hunt,[2] Cecil and Steffen[3] and Moore.[4] Cutler and Hunt are agreed with Fetterolf and Fox that

[1] Fetterolf, George, and Fox, Herbert: Reaction of Peritonsillar Tissue to Tonsillectomy. (A Study of the Etiology of Posttonsillectomy Lung Complications.) In print.

[2] Cutler, Elliott C., and Hunt, Alice M., R.N.: Arch. Int. Med., April, 1922, p. 449.

[3] Cecil, Russell L., and Steffen, Gustave I.: Jour. Exp. Med., August 1923, p. 149.

[4] Moore, Wm. F.: Pulmonary Abscess (An Analysis of 202 Cases Following Operative Work about the Upper Respiratory Passages), Jour. Am. Med. Assn., 1922, lxxviii, 1279.

in a larger number of these cases than have previously been so credited the infection follows the blood-stream route to an ultimate termination in the lung tissues.

We do not assume the burden of the proof, but would like to point out certain conditions, factors and observations which have led us to place aspiratory infection as the most common cause.

Moore, after an analysis of the localization of the suppurating area in 202 cases which showed that the lower lobes were more often involved (41 per cent right and 19 per cent left), thought it justifiable to assume that the infection had followed the same route as for aspirated bronchial foreign bodies (Jackson[1]), which show in their localization approximately these percentages. The article by Fetterolf and Fox states that the lower lobes are the ones most commonly affected from a blood-stream infection. Their contention, however, that infarction usually takes place, of which one of the cardial early symptoms is expectoration of bright red blood, which they say may often be confused with secondary hemorrhage following tonsillectomy, has not been borne out by the history records of the cases of posttonsillectomic lung suppuration that have come to the Bronchoscopic Clinic.

It is an undisputed fact that the vast majority of postoperative cases of pulmonary abscess follow operations about the upper respiratory tract. Surely the same conditions pertain to any surgical procedure about the body as far as the blood-stream is concerned, whereas the aspiratory factor only becomes of importance in this connection when working about the upper respiratory tract. Fetterolf and Fox observe that "The actively muscular pharynx is a particularly favorable region for the dislodgment of such thrombi into the superior caval circulation," but surely abdominal operations have this same complication, due to respiration and peristalsis, and a patient with a raw surface in the throat swallows as little as possible.

Considering the number of such operations done at the present time, it is pertinent to inquire why this complication does not

[1] Jackson, Chevalier: Peroral Endoscopy, Laryngoscope Co., 1914.

occur more often than is reported. Careful inquiry since the work of Richardson, Scudder, Manges and others, some thirteen years ago, has brought to light pulmonary suppurations as a complication in increasing numbers. Making due allowance for the cases overlooked because of a lack of a "follow-up" system, the complication does not seem, relatively, a frequent one.

With either mode of infection would it not be well to take into consideration as explaining its comparative rarity the question of individual immunity derived from chronic infections of the nose and throat? For example, a person infected with chronically diseased tonsils should certainly have developed a degree of resistance to the bacterial growth which is conveyed to the lungs at the time of operation. This question then leads naturally to others. It would seem, after a study of the cases which have come for treatment, that a patient either in poor physical condition or in extreme shock at the time of operation had overcome the barriers of natural resistance and developed a field where infection plus lowered resistance resulted in pulmonary suppuration.

The work of Cecil and Steffen is interesting in this connection, in that they were able to produce pneumonias in control monkeys by intratracheal insufflation of rich pneumococci cultures. These occurred in the lower lobes and more frequently in the right lower lobe in animals that had not previously been immunized.

Lynah[1] inferred from roentgenograms taken after injections of bismuth suspensions made through a bronchoscope that the actual abscess cavity was usually smaller than had previously been supposed. Opinion at the Bronchoscopic Clinic is that the reverse is the case and that involved areas are really larger than indicated by opaque mixtures because the so-called cavity is filled with granulations that prevent the opaque mixture from extending fully out to the periphery of the suppurating area.

Treatment. The treatment of suppurative conditions of the lungs, and especially pulmonary abscess, has been attempted in many ways. These conditions, as is the case with many other

[1] Lynah, Henry L.: Lung-mapping by Injections of Bismuth Mixtures in the Living, New York Med. Jour., 1921, cxiv, 82.

TABULATED RESULTS OF BRONCHOSCOPIC TREATMENT IN CASES OF PULMONARY SUPPURATION

No.	Sex.	Age.	Duration of disease.	Probable cause.	First symptom.	Location of lesion.	Amount of sputum.		Fetor.		Number of bronchoscopies.	Present status.
							Before treatment.	After treatment.	Before treatment.	Present time.		
1	F.	26	23 yrs.	Measles	Productive cough	Pul. sup. R.L.L.	.....	.....	Present	Absent	33	Cured.
2	M.	23	21 yrs.	Unknown; measles (?)	Productive cough	Pul. abscess R.L.L.	.....	.....	....	....	16	Improved.
3	F.	34	1 yr. 7 mos.	Severe pharyngitis; laryngitis	Dry cough; productive 6 mos. later	Pul. abscess and bronchiectasis left lung	Large amount	Reduced one-half	Present	Decrease	11	Temporary improvement.
5	M.	7	7 yrs.	Unknown	Cough at 2 weeks. Later productive	L.L.L. R.L.L.	Enormous amounts	Decreased	Slight	Absent	40	Improved.
12	F.	21	2 mos.	Tonsillectomy	Productive cough	Pul. abscess R.L.L.	¼ cup	Absent	None	None	16	Cured.
14	F.	49	8½ yrs.	Tooth extracted; foreign body	Cough; later productive; malaise	Pul. abscess R.L.L.	Great quantity	Decreased	Present	No change	5	Slight improvement.
21	M.	12	8 mos.	Tonsillectomy	Pain in post. rt. chest followed by cough; productive	Pul. abscess R.L.L.	Large amounts	¼ cup	Slight	Slight	9	Improved.
27	M.	16	1 yr. 3 mos.	Tonsillectomy	Pneumonia (?) cough	Pul. abscess R.L.L.	Large amounts	Decreased	Present	Lessened	12	Improved.
29	M.	40	23 days	Unknown	Fever; chills; productive cough; hemoptysis 17 days later	Pul. abscess L.U.L.	Large amount	Decreased	Present	None	9	Improved.
30	F.	27	11 mos.	Cholecystitis; ether anesthesia	Recurrent cough	Pul. abscess R.U.L.	Large amounts	Occasional small amounts	Present	Absent	9	Marked improvement.
35	F.	23	6 wks. 2 days	Tonsillectomy	Fever; cough; expectoration	Pul. abscess R.L.L.	Large amount	Small amount occasional	Present	Absent	3	Cured.
38	M.	46	8 yrs.	Unknown	Cough; productive	Pul. abscess L.L.L.	Above 40 oz.	27 oz.	Present	Decreased one-half	8	Improved.
41	F.	38	3 yrs.	Childbirth ether anesthesia	Cough; later productive	Pul. abscess L.L.L.	1000 cc	250 cc	Present	Less	6	Improved.

ailments to which mankind is subject, either progress to spontaneous recovery due to factors, chief among which is the fortunate rupture of the affected area into a bronchus patulous enough effectually to drain the abscess, or the condition becomes chronic with the definite involvement of a portion of the lung lobe surrounded by an area of edematous lung tissue, and because the condition is so well described by the words "sponge soaked;" this term has been well used to define that tissue immediately surrounding the abscess.

The treatments advocated for this condition include:

1. Confinement to bed for long periods of time with an abundant nutritious diet.[1]
2. Postural treatment.
3. Vaccine treatment.
4. Bronchoscopic aspiration and irrigation or the instillation of medicaments.
5. Surgical procedures, which include:
   (*a*) Artificial pneumothorax.
   (*b*) Phrenectomy.
   (*c*) Ligation of the pulmonary artery.
   (*d*) Thoracoplasty.
   (*e*) Lobectomy.

Bronchoscopic treatment has now progressed to such a point that hope can be held out to the patient of decided amelioration of the distressing symptoms, and certainly in an as yet undetermined percentage of those cases unaffected with a generalized bronchiectasis of a permanent cure. In any case the patient has lost nothing through delay while undergoing bronchoscopic treatment, and indeed a localization or a limitation, or both, may influence for good the end-result. Moreover, he has incurred practically no risk by bronchoscopic treatment.

After treating many of these cases we have concluded that the use of one method to the exclusion of all others is a mistake. Therefore, routinely, when a patient with a pulmonary suppuration

[1] Pritchard, J. S.: The Value of Rest Cases of Empyema and Lung Abscess, Jour. Am. Med. Assn., December, 1922, p. 2208.

presents himself or herself at the clinic the following procedures are instituted:

The chest findings are ascertained by an internist, who also makes a thorough study of the patient for organic disease, after which the patient is referred for roentgen-ray examination. Tuberculosis is excluded so far as possible by the usual diagnostic means. With the information thus obtained it then becomes a question of whether or not the case is one for a diagnostic bronchoscopy.

This preliminary work is necessary not because the bronchoscopic procedure is in any way dangerous, in proper hands, in a fit subject; but suitability of the case for bronchoscopic examination and treatment must be determined on the broadest scientific principles, not only for the best interests of the particular case under observation but for the development to the fullest extent of criteria by which suitability can be determined in future cases. For instance, we recall a case in the past year sent from a distant city for bronchoscopy which on examination proved to be complicated by an extensive empyema. We did no bronchoscopy because the case was utterly unsuited to bronchoscopy, requiring immediate aspiration through a pleural external opening. The pleural cavity cannot be adequately drained by oral bronchoscopy, though intercostal pleuroscopy is often helpful. In this case even external surgery was unable to relieve a general septic condition, which cost the patient's life. Had we done a bronchoscopy we could have done the patient no good and would have brought undeserved discredit on a procedure that is of life-saving importance in properly selected cases.

Having localized, so far as possible, an abscess or bronchiectatic condition, a diagnostic bronchoscopy is performed. The condition of the mucous membrane of the tracheobronchial tree is noted as the bronchoscope is inserted. Two of the most important points to be carefully watched for are these:

1. The bronchus or bronchi from which suppurative products are exuding.

2. The presence of any factor which would constrict the lumen of the bronchus and thus inhibit free drainage.

At this examination uncontaminated specimens of the secretion are taken either by swab through the bronchoscope or, if the quantity is sufficient and not too viscid, the collection is made by aspiration into a specially devised tube,[1] which can immediately be sealed and sent to the laboratory.

From these specimens, uncontaminated by oral infections, autogenous vaccines are made in each case. These vaccines are given twice a week. Fresh vaccines are made up at two-month intervals, because we have found that the predominant bacterial content changes in patients under treatment. This change may be due in part to the treatment.

These patients are carefully instructed to aid by posture the drainage of the affected area; that is, by assuming a sleeping position on the right side if the left is affected and *vice versa*, and also by observing special instructions as to posture during waking hours.

We have never insisted on absolute bed rest unless there is fever or weakness, but an abundant, nutritious diet, as for tuberculous patients, is outlined. Fatigue must be avoided and rest in bed a definite number of hours, (fourteen to eighteen) each day, to conserve strength and to build up a reserve is recommended.

At the second bronchoscopic treatment the bronchi draining the affected area are first thoroughly aspirated, using either the Jackson aspirating bronchoscope or, in case of the small bronchi whose lumen will not take the scope, using an independent aspirating tube inserted through the bronchoscope. The cleansing is with a special solution containing:

Trinitrophenol	2 gr.
Lugol's solution	ʒj
Normal saline solution	1 pint

This completes the treatment in some cases. Seven-day intervals are usually allowed between bronchoscopies.

[1] Lukens, R. M., Moore, W. F., Funk, E. H.: Bronchoscopic Drainage of Pulmonary Abscesses and Bronchiectasis, Clinics of North America, January, 1923, p. 1015.

In cleansing, large areas are never treated at one time, and flooding of healthy lung tissue is carefully avoided. The tributary branch bronchus is attached separately, excluding the main trunk, and with specially devised apparatus. The return flow is always greater than the intake. A washing action is thus secured, and at the end of the treatment very little fluid is retained. This guards against a scattering of the infection to normal areas. Vegetable oils have been found very unsatisfactory. Only mineral oils are used. These are at times used either after irrigation or instilled after aspiration without any irrigation. We have yet to see a case where infection was spread after this treatment.

The lumen of a constricted bronchus is dilated at each bronchoscopy. Obstructing granulations sometimes require removal, but usually granulations disappear after purulent accumulations are prevented by bronchoscopic aspirations.

Bronchoscopy is done without anesthesia or sedative in children, and without general anesthesia in adults.

These patients are so little affected by this procedure that they are able to leave the clinic for their homes almost immediately and are treated as dispensary patients. Hospitalization is not required, though sometimes out-of-town patients are put in the hospital for preliminary study.

Results. An analysis of the cases of pulmonary abscess treated at this clinic, including those complicated with bronchiectasis in percentages, is as follows:

Cured	21.4 per cent.
Improved	57.2 "
Unimproved	21.4 "

The cured cases are pronounced cured only after prolonged cessation of symptoms and complete *x*-ray studies.

The improved cases have had marked amelioration of symptoms, and in most instances have been able to resume occupations. The improved cases include those on whom we are still working, hoping for an ultimate cure.

The unimproved class was chiefly made up of those patients

with extensive bronchiectasis. These cases, as before mentioned, respond very poorly or not at all to this treatment.

Patients are willing to continue the treatments as long as we deem it advisable, because of the lessening of cough, foul expectoration and improvement. We have yet to see the patient who declined further treatment because of any dread of a repetition of the bronchoscopic treatment.

To the often-asked question, "In what class of cases do you believe bronchoscopic treatment indicated?" our reply has been that, while this question can be definitely answered only after many thousands of cases have been treated, in a general way it may be stated that as a working basis for the present we believe diagnostic bronchoscopy is indicated:

(*a*) In any patient in good general condition and with a not too extensively diseased lung.

(*b*) Any case in which the internist desires the aid of bronchoscopy with aspiration or medication as an addition to his general measures.

Bronchoscopy is contraindicated.

(*a*) In cases of recent profuse hemorrhage.

(*b*) In a moribund patient.

(*c*) In very extensive disease of lung tissue, involving one-half or more of one lung.

(*d*) In organic disease of the heart and great vessels.

(*e*) In laryngeal tuberculosis.

Asthma. We are now at work on a large group of cases of bronchial asthma. It is too soon for definite conclusions, but brief citation of cases showing mode of treatment and results after six months is included.

An autogenous vaccine is made at the first bronchoscopy, then a medicated solution is instilled first in the right and then in the left main stem bronchus. The following is the formula we have most frequently used:

20 drops 1 to 1000 adrenalin solution.
10 drops 10 per cent cocaine.
Normal saline, ʒj.

Some of these cases have a considerable quantity of thick, tenacious secretion clining to the bronchial walls. This should be removed before instillation of the special formula either by suction or swabbing.

These treatments were given at seven-day intervals generally for the first three times, after which the patient reported not oftener than once every three weeks or a month. However, in each case they reported only on recurrence of asthma.

The bacteriological study of bronchoscopically secured specimens from patients with tracheobronchial asthma has not been completed. Generally staphylococci, streptococci and catarrhalis were present in cultures.

The mucous membrane at the first bronchoscopic examination generally presents a bluish-red appearance, as of a passive congestion. At the second treatment this is not so marked, and has generally disappeared at the third bronchoscopy. In 2 cases there was almost complete expiratory collapse of the bronchi and trachea. One case seemed to collapse anterioposteriorly and one laterally. In several cases the asthma was accompanied by a real tracheobronchitis. No spirals or puriform bacilli have been found. Charco-Leyden crystals were reported in only 1 case.

Results in Asthmatic Cases. Sixteen cases have been treated. Fifty per cent of these have showed marked improvement. One case reported recently for the first time in three months, stating he had been free of asthma until the day before reporting, and that this was his longest respite in seven years. Before treatments he had had attacks daily. He had been given seven treatments. Another case was complicated with pulmonary abscess. Her doctor reports that she is now pregnant, and that the lung symptoms are giving her no trouble.

Another case received ten treatments, and is now able to resume an occupation which previously was interrupted by his attacks of asthma.

In all, 16 cases have received treatment, of which we can report a definite improvement extending over three months in 5. Of the others, 3 are questionable, 1 having received only one and the others two treatments, and have not since reported to the clinic.

Conclusions. 1. Aspiration of infective material is an important factor in the production of postoperative pulmonary abscess.

2. Bronchoscopy is attended with no danger when performed by a team properly trained to work together.

3. Bronchoscopy is the logical means to facilitate better drainage and aid medical treatment. Free drainage is always fundamental for suppuration anywhere.

4. Patients are willing to continue the treatments as long as we deem it advisable, because of the subjective evidence of the lessening of cough and foul expectoration and of the improvement in the general health.

5. Cases of pulmonary suppuration cannot all be cured by bronchoscopic means. A certain percentage certainly can be, and a large percentage can be improved. The rest, with few exceptions, are made happier through the alleviation of symptoms which have made the patient thoroughly wretched both day and night.

6. Bronchoscopic instillation of medicated solutions seems to alleviate "asthmatic attacks" for relatively long periods.

Studies of Mastoid Disease by the *X*-rays, with Clinical Observations and Operative Findings[1]

by Matthew S. Ersner, M.D.

It is a pleasure and honor to be here tonight to collaborate with Dr. George E. Pfahler on the subject chosen for us this evening.

In preparing this paper, I have tried to present the subject in as practical a form as possible, and this alone, I hope, will suffice to convey a crystallized idea of the subject.

I have studied in conjunction with Dr. Pfahler 142 mastoid cases. In this little bundle of records there is a store of knowledge

[1] Presented by invitation in conjunction with *x*-ray studies by G. E. Pfahler, M.D. Illustrated by lantern slides.

which in quiet moments I often review. The x-ray is a study of dead shadows of varying densities, which to the casual onlooker means nothing, but to the donors and clinicians the plates recall trying moments, aching pains and sleepless nights, etc. So when I look at the plates there appears through each the hazy image of the patient. From our collection we have selected 19 slides, representing various typical and atypical interesting acute mastoid types. It is our routine to order x-ray examinations for every mastoid patient, whether private, semiprivate or ward, irrespective of the clinical diagnosis, whether conclusive or not. Some think this procedure extravagant, others forget the x-ray when the clinical findings point toward operation. I, too, was skeptical at first in my inclination toward the x-ray in mastoid disease, but have since learned its indispensability.

Many men usually x-ray their atypical cases only, which under such circumstances we may have atypical anatomical abnormalities to deal with, and the shadows may therefore be misunderstood. The x-ray is the sequence of a physical, mechanical and chemical procedure, accomplished by the penetration of the rays through the suspicious area and registering their course by casting various shadows. I believe that every shadow is a message for us to interpret. These may be helpful, misleading or misunderstood. It should be our aim in life to have the most knowledge in the field of our daily pursuit, and since knowledge is based upon experience accumulated, one should therefore, in this instance, study as many plates as possible and correlate the various shadows with the clinical and operative findings. The x-ray plates to me are less puzzling daily, and I feel that I am having less difficulty in interpreting them. I often see shadows which recall cases similar in nature which heretofore were Chinese puzzles to me, but now are concrete facts without pitfalls.

What should we as clinicians expect from the x-ray in mastoid disease?

1. Comparative study between the normal and the diseased mastoid. (Both mastoids are usually developed alike and any deviation from the normal will be divulged by the x-ray plate.)

2. Type of mastoid, whether pneumatic, non-cellular or sclerotic.

3. Topographical and regional anatomy of the mastoid, its cellular distribution, type of cells, position of the sinus and its surrounding area.

4. In atypical mastoiditis the *x*-ray is indispensable as an aid in diagnosis. (See Case VI–R.)

5. Cavity formation.

6. Prognosis is often made by consecutive *x*-ray studies. (A mastoid with large cells may undergo resolution without operative interference, as the drainage in such mastoid is not impeded as frequently as in the other mastoid types.) (See Case III–T.)

7. Diagnosis of perisinus abscess. (See Case XIII–C.)

8. Epidural abscess diagnosed. (See Case VI–R.)

9. Bilateral suppuration. (Cases XVI and XVIII–F and G.)

10. Sclerotic mastoid. (Case XVII–S.)

11. In children where the cells are not fully developed a roentgenological diagnosis may often be made when the antrum is located, as there are a few cells invariably present there.

Let us visualize hypothetically, step by step, the pathological changes taking place in the mastoid and interpret these phenomena on the *x*-ray plates, providing these are pneumatic mastoids. In an acute mastoid of recent origin we at first have hyperemia and the congestion is the same as in any other part of the body. Roentgenological studies of both mastoids reveal that the normal side is clear and that the cellular components stand out sharply with distinct cellular septa, while in the congested side all the normal mastoid elements are present, but in addition there is a slight haziness appearing as though there were a slight error in focussing. (See Case VI–R.)

In this stage either resolution takes place or the pathology continues. We may assume that the bacteria here are active and that the mucous membranes lining the mastoid cells are becoming more edematous. We also have a constant infiltration of round cells and an increase in the leukocytes, the congested area gradually becomes purulent. The *x*-ray plates at this time will reveal marked

cloudiness with a tendency to obliteration of the cellular spaces appearing as though they were almost filled to the brim. The cellular septa are still visible as a minute network of fibrillated strands, and our interpretation of the plates at this stage should be exudation present. In the advent of an increase in the purulent constituency there is also an increase in the intracellular septa. The picture now presents a distinct opacity with a blurring of the cellular septa. In case the surgeon decides to follow the conservative course of watchful waiting the lateral sinus under these conditions should be outlined and studied carefully in the consecutive pictures. When the lateral sinus is lost we may suspect a perisinus abscess with a loss of the bony structure covering the anterior bony sinus wall. Operation under these circumstances is imperative and should be done immediately. Absorption, liquefaction and cavity formation appear on the *x*-ray plate as a dark spot or as a shadow more or less regularly outlined with the edges blurring and no trabecula visible. (See Case XVII.) In contradistinction the normal large cell is irregular with the edges sharply outlined, and the cellular septa are distinctly visible. A loss of cellular structures over the zygomatic area with an extension of the haziness toward the squamous portion of the temporal bone is suggestive of an epidural abscess (as in Case VI).

In the 19 cases presented here we have a detailed study, including the history, clinical and operative findings, which are correlated with the roentgen studies.

Case I.—Baby R., aged eighteen months. (February 5, 1923.)

History. Baby is a child of a physician whose mother and brother were sick with grip. As they were recuperating this child fell a victim. The temperature fluctuated for six days, when I was called to see the baby, and I found a bulging right ear-drum. Incision of ear-drum with flow of free pus. For four days it looked as though all was well, when there was a recrudescence of the symptoms. The ear-drum was reincised five times, but the pus continued to discharge profusely. A pediatrician was called in during my absence and he incised the other ear-drum. The middle

ear in this instance became infected, but the drainage was not very profuse, and about ten swabbings daily sufficed to keep the middle ear clean. After several weeks of bilateral suppuration the clinical signs and symptoms centered upon the right ear. The *x*-ray showed either destruction or undeveloped mastoid cells.

Laboratory Findings. Leukocyte count, 14,000. Urine analysis, negative. There was also a secondary anemia present, poikilocytes, anisocytes and nucleated red blood cells.

X-ray Report. 1. Mastoid unusually large for a child of eighteen months. Disease apparently involves the entire area. About 4 cm. in diameter. There are transparent areas in the zygomatic region, which are probably also decalcified trabeculæ. (Diseased area marked with arrows.)

On March 6, 1923, I thought I felt a thickening of the periosteum, and on March 9 there appeared a slight displacement of the auricle.

Operative Findings.

1. Periosteum slightly thickened and not adherent.
2. Cortex thin.
3. Small pin-point perforation of cortex opposite the spine or Henle (not developed).
4. Pus under pressure.
5. Small cavity at tip and extensive necrosis of the zygomatic cells. The rest of the mastoid cavity was filled with pus, granulation tissue and in discrete areas; the cellular elements were undisturbed.
6. The mastoid cavity was very large and the antrum very deep.

Case II.—F. R., aged six years. (April 9, 1923.)

History. Patient had measles, which was complicated by an acute suppurative otitis media. The ear-drum on the right side ruptured spontaneously, and when seen there was a profuse discharge. The temperature flucutated between 99° and 103° daily, with severe pain over the right mastoid. The other general symptoms, such as restlessness and insomnia, accompanied.

Laboratory Examination. Leukocyte count, 16,000. Urine negative.

Physical Examination. (*a*) Profuse aural discharge.

(*b*) Pus under pressure.

(*c*) Sagging of posterior-superior canal wall.

(*d*) Impaired hearing.

(*e*) Loss of postauricular sulci, only one-half fold remaining while three and one-half folds were present on the good side.

(*f*) On palpation there was a slight thickening of the periosteum, and on deep pressure I located two pronounced tender spots, one at the posterior portion of the tip and the other at the distal end of the zygomatic ridge.

X-ray Report. Mastoid area comparatively small. The mastoids belong to the small pneumatic type. The left side is more fully developed than the right. There is evidence of disease, with absorption of lime salts in the regions of the knee of the lateral sinus and of the tip. The sinus walls are well marked. (Diseased area marked with arrows.)

Operative Findings. 1. Periosteum thickened and stripped with difficulty.

2. Cortex congested with slight greenish discoloration at the tip.

3. Cortex thick.

4. Pus thick and creamy but not under tension.

5. Two cavities were present, one at the tip and the other at the knee of the lateral sinus.

6. The zygomatic cells were partly destroyed, but the rest of the mastoid cells were filled with granulation tissue and pus; the cells were soft but not liquefied.

Case III.—Mrs. T., aged forty-five years. (April 6, 1923.)

History. Patient had an acute suppurative otitis media, which was a complication of grip. She was first seen in my office March 24, 1923, complaining of pain in the ear, with tinnitus and impaired hearing.

Examination. A bulging ear-drum was revealed, which I incised and free pus was found. Four days later (March 28, 1923)

she developed pain over the mastoid, with temperature ranging around 100°, with no abatement of the symptoms. On April 3, 1923, the discharge from the ear became more profuse. She was sent into the hospital for observation, where she was *x*-rayed. Her condition cleared up after being in bed for one week.

X-ray Report. Mastoid is of the large-cell pneumatic type. The left is more extensive than the right. The right cells are clouded throughout. (Diseased area marked with arrows.)

Conclusion. This mastoid was of the large pneumatic type, and by putting the patient to bed it gave her a chance to fight the infection off and the large cells drained themselves and spontaneous cure resulted.

Case IV.—L. G., aged five and a half years. (September 22, 1923.)

History. Patient had measles complicated by an acute suppurative otitis media. When first seen in the office she gave a history of a discharging ear three weeks prior, with spontaneous rupture of the ear-drum. The discharge lasted for nine days after, when the ear became dry. Mother noticed that there was a complete loss of hearing in the right ear, and this was verified during conversation. The child's chief complaint was pain at night and pain upon manipulation.

Examination. Child was very intelligent and coöperative.

(*a*) Impaired hearing with complete loss of whispered voice and only five feet of spoken voice.

(*b*) Fork Tests: Loss of low tone, Rinné negative; Weber lateralized to the right; Schwabach plus.

(*c*) Ear-drum apparently normal.

(*d*) Inspection. Loss of postauricular sulci while normal side had three folds.

(*e*) Thickening of periosteum over mastoid.

(*f*) Slight suggestion of displacement of auricle.

(*g*) Pain elicited only on deep palpation in the following areas: (1) Over the emmisary vein; (2) over the tip; (3) over the antrum.

X-ray Report. Mastoid pneumatic in type, large cells. Local bone absorption at the tip, with general clouding of the remainder of the cells. (Diseased area marked with arrows.)

Operative Findings. 1. Periosteum slightly thickened but not adherent.

2. Pin-point perforation opposite the spine of Henle.

3. Cortex of medium thickness.

4. Cells large.

5. Cavity at the tip and small cavity opposite the spine of Henle. The rest of the cells were soft and filled with exudate, but not broken down.

No temperature present during the study of this case.

Case V.—A. S., aged nineteen years. (December 23, 1922.)

History. Patient had grip ten days ago and three days later the nasal discharge became thick and profuse. On this day she sniffled some salt solution, so as to wash out nose, when she felt a click in the right ear and pain ensued.

Examination. 1. The patient looked toxic, sordid lips, dry, coated tongue, eyes cloudy, nose stuffed and repeated blowing.

2. Bulging ear-drum was incised under ethyl-chloride anesthesia and pus was present.

3. Palpation. Pain all over mastoid upon light manipulation.

4. Hearing tests. Weber lateralized to the right; Rinné negative; Schwabach plus.

The discharge continued for eleven days without cessation. The pain became unbearable, hearing almost completely lost. Whispered voice not heard at all and spoken voice only heard within two feet.

Laboratory Findings. Leukocyte count, 14,000; urine negative.

At this time the periosteum felt thicker with a loss of the post-auricular sulci. Tenderness all over the mastoid.

X-ray Report. Pneumatic type. Large activity extending from the knee of the lateral sinus to the tip and extending back over the lateral sinus. Anterior wall of the sinus cannot be seen. (Diseased area marked with arrows.)

Operative Findings. 1. Periosteum thickened, adherent and stripped with difficulty.

2. Cortex very thick.

3. Large cavity at the tip with some absorption around the zygomatic area.

4. Cell extension was marked and all cells were filled with granulation tissue, with a small cavity posterior to the lateral sinus. The lateral sinus was exposed and covered with granulation tissue, which was not disturbed.

Case VI.—D. R., aged seventy-three years. (March 19, 1923.)

History. Patient was a hard-working, sturdy farmer. Three months prior to examination he gives an indefinite history of having had pain in the right ear. He was seen by a local physician. At no time did he have a discharging ear. He suffered pain for about four weeks before he came to the city to seek advice and relief. He was seen by a general practitioner. Neurologic examination was negative. Urine negative. Blood-pressure normal. No evidence of optic atrophy. His chief complaint was pain over the squamous portion of the temporal bone. He was shaved and leeches were applied for two days. He was relieved of pain for several days, but the pain returned more severely. It was then I was called in consultation.

Examination. 1. Severe, continuous headache over right side, especially over squamous portion of the temporal bone.

2. Patient felt as though some one were pulling constantly at his head.

3. Ear-drum negative.

4. Hearing tests: Loss of low fork; loss of whispered voice; Weber lateralized to the right; Rinné negative; Schwabach increased; MacKenzie plus.

There was a slight postauricular edema caused by the leeches. Inspection, therefore, was of no value. There was severe pain on deep palpation over the zygomatic and squamous portion of the temporal bone.

Laboratory Findings. Negative.

X-ray Report. Mastoid of the pneumatic type extensive in area on the normal side (left) clearly defined. On the right side the mastoid area shades off into the surrounding tissues, as though the surrounding bone had been partially absorbed. The trabeculæ can be recognized at places and at others seemed to be totally lost. (Diseased area marked with arrows.)

Operative Findings. 1. Periosteum thickened and adherent.

2. Cortex very thick.

3. Cells very large and pus under pressure and pulsating.

4. The whole mastoid cavity was a mass of liquefied tissue.

5. The sinus was completely exposed and covered with granulation tissue. There was also an extradural abscess in the middle fossa.

I was unable to explain the shadow around the parietal region which I saw on the *x*-ray plates, as the bone looked healthy and I did not follow that course through, unless the shadow was due to the pus pocket, which emptied itself when the cortex was removed.

6. The necrosis extended about ½ inch posterior to the lateral sinus.

7. The mastoid cavity was very deep and wide.

Operative. Local anesthesia, 1 per cent novocaine, and 1½ ounces were used. (This was the prize case of the year. It was a classic.)

Case VII.—T. S., aged twenty-three years. (March 9, 1923.)

History. Patient was sick with grip and after four days developed acute suppurative otitis media as a complication. He developed severe pain in the right ear, which became progressively worse.

Examination. 1. Nasal congestion and profuse thick nasal discharge.

2. *Ear Examination.* Found three distinct discolored bluish blebs in different parts of the ear-drum. These were punctured and fluid trickled out, after which the ear-drum came into view and was found to be sagging, and when incised there was a flow of free pus The ear-drum was re-incised at different intervals so

as to promote drainage, but there was no abatement of the symptoms. This continued for about three and a half weeks, when the sharp, lancinating pains were displaced by a hemicrania, a loss of hearing and a heavy boring pain. His general condition became anoying but not alarming.

3. *Chest examination* revealed fibrosis, with some activity, although sputum was negative for tuberculosis.

4. Examination on day of operation. Profuse aural discharge, which was pulsating. Sagging of posterior-superior canal wall.

5. Fork tests: Loss of whispered sound; loss of low tone fork; spoken voice audible at 7 feet; Weber lateralized to the right; Rinné negative; Schwabach plus; MacKenzie paradox positive for four seconds.

Temperature. Normal.

Laboratory Tests. Leukocyte count 11,200. Urine analysis negative.

Inspection. Loss of postauricular sulci. No apparent displacement of the auricle.

Palpation. Periosteum over mastoid thickened and seemed adherent while on the normal side the skin was readily movable.

Superficial Palpation. No pain.

Deep Palpation. Pain over tip and pain at posterior edge of mastoid at the point of the emissary vein.

X-ray Report. Mastoid of the pneumatic type, large cells, showing destruction about the knee of the lateral sinus and also extending posteriorly to the lateral sinus. (Diseased area marked with arrows.)

Operative Findings. 1. Periosteum thick and adherent.

2. Cortex thick.

3. Superficial layer of cells of medium size slightly congested but no liquefaction present.

4. Second layer of cells filled with pus and granulation tissue was found only in certain areas. There was a tendency to coalescence.

5. Cavity at the tip.

6. Perisinus abscess and anterior bony wall of sinus completely gone, with a large cavity posterior to the sinus.

7. Sinus covered with a bluish granulation tissue, which was not disturbed.

8. Pus found in these cavities was thick but not under pressure.

Conclusion. In this mastoid there seemed to be three strata of cells. The upper strata was almost healthy, the second strata was filled with pus and granulation tissue, while the floor of the mastoid had all the pathology. This mastoid was deep, very wide and the cellular distribution was extensive.

Local anesthesia, novocaine, 1 per cent, and 1½ ounces were used.

CASE VIII.—S. S., aged sixteen months (July 9, 1923.)

History. Patient had acute rhinitis, which terminated in acute suppurative otitis media, bilateral. Both ears were incised and free pus found. The discharge and toxic temperature lasted for six weeks.

Laboratory Findings. Leukocyte count, 22,000, 28,000, 21,500. Urine analysis, faint trace of albumin.

Examination on Day of Operation. 1. Profuse aural discharge and pus under tension.

2. Sagging of the posterior-superior canal walls.

3. Restlessness and constant crying.

4. Postauricular tenderness all over the mastoid. More marked on the left side, especially over the zygomatic area.

X-ray Report. Mastoid poorly developed, though normal for age. Lime absorption in both sides in the region of the antrum, more on the left than on the right. (Diseased area marked with arrows.)

Operative Findings. 1. Bilateral mastoidectomy performed.

2. Periosteum thickened and somewhat difficult to strip.

3. Cortex looked good and of medium thickness.

4. Pus thick and under pressure.

5. Discrete areas of liquefaction.

6. Dehiscence of dural plate about the size of a dime (left side).

7. Mastoid cavity very deep, about 1 inch in length and $\frac{3}{4}$ inch wide.

8. Both antra were deep and extensive necrosis was found around the antra.

Case IX.—L. P., aged fifteen months. (March 18, 1922.)

History. This child, when first seen, had a temperature of 104°. She was very restless and looked ill. There was a history of intestinal disturbance, with flatulence. One week previous to this date the baby was seen by a pediatrician who incised the right ear drum, but there was no discharge. Five days later the left ear opened spontaneously. Examination of the ear revealed a sagging in the posterior-superior canal wall of the left ear, with profuse discharge. The right ear-drum revealed a linear incision, with a thin serous discharge. I watched the baby for four days consecutively without abatement of the symptoms. On March 22, 1922, a postauricular examination disclosed 1 crease on the left side and $2\frac{1}{2}$ creases on the right side. At this time I found that there was a slight thickening of the periosteum and immobility of the skin over the left mastoid. I sent the baby into the hospital and ordered both ears *x*-rayed. The picture showed the semicircular canals *in situ*. The leukocyte count was 25,000. The mastoids were rephotographed three times, and at no time could we make a definite diagnosis from the *x*-ray readings. I did, however, note that on palpation, especially on deep pressure over the left mastoid, the child made an attempt to pull my hands away, and her cries and contortions became more aggravated. On March 25, 1922, although at this time there was some slight tenderness over the right mastoid, I decided to open the left mastoid and found it to be completely broken down. The temperature subsided, and on the fourth day it reached normal. On the eighth day I found both ears completely dry. The baby was discharged from the hospital April 5, 1922. On April 15 I again was called to see the baby, who was being attended by a pediatrician for the past five days, treating the intestinal tract. The same history of intestinal

disturbance with flatulence was given, and all our efforts were concentrated toward the intestinal tract.

Examination of Both Ears. I found the left ear dry and the mastoid wound clean. The right ear revealed a scar from the previous incision.

Postauricular Examination. There was no displacement of the auricle, but there was a smoothing out of the folds, leaving only one-half of the postauricular fold intact. I saw no urgency to return the baby to the hospital, as our *x*-rays were negative. We did, however, find some tenderness over the mastoid, especially on deep palpation over the antrum and at the tip. The leukocyte count at this time was 15,000 and the differential was 80 per cent polymorphonuclears. With a low leukocyte count and high differential cell count, and with a history of previous suppuration and sudden cessation, a loss of postauricular creases, tenderness on deep pressure, and with the previous hazy picture of intestinal disturbance, I felt that we should not lose sight of the fact that the right mastoid might be involved. On April 20, 1922, I ordered another *x*-ray of the right mastoid. The cellular structures looked blurred, but there was no evidence of any breaking down. There was no history of any chills, but the temperature continued to range between 100° and 104°. On April 26, I again met the pediatrician, and we went over the case together, and after a long conversation, we decided that the flatulence, although it persisted, was not enough to explain the toxic symptoms that the child presented. We decided to operate on the right mastoid. The leukocyte count was 15,000 and the polymorphonuclear was now 80 per cent. A postauricular incision was made and I found the periosteum thickened and adherent. There was a thick, cortical layer which I removed. I found the pus under tension and the mastoid completely broken down. The baby made an uneventful recovery. This patient taught me a lesson that the clinical picture should stand out primarily.

X-ray Report. Mastoid cells undeveloped. No evidence of disease shown. Semicircular canals clearly shown. (Diseased area marked with arrows.)

Note. The semicircular canals were visible in the plate, because in children there is very little lime salt and the rays therefore penetrate through everything until they reach the hard, bony structure.

Conclusion. The clinical picture is an important factor to be borne in mind, as you will note the *x*-ray findings do not coincide with the clinical and operative findings.

CASE X.—S. P., aged eleven years. (January 23, 1923.)

History. Patient had rhinitis for several days and after forceful blowing of the nose developed pain in the left ear. He presented himself at the office January 23, 1923, with a profuse aural discharge from the left ear-drum. During the interval between January 23 and February 22 I only saw him twice. On February 22 the discharge became very thick and lessened. On February 26 the discharge practically ceased, but the pain over the mastoid increased. Upon examination I found that there was only a small linear perforation in the ear-drum with thick pus exuding. There was a loss of the postauricular sulci and thickening of the periosteum. On February 27 auricular displacement was noted and the tenderness over the mastoid became more marked. His hearing was impaired and he at no time had any temperature.

Laboratory Findings. Leukocyte, 11,200. Urine negative.

X-ray Report. Mastoid on the right side, large, pneumatic in type, with medium-size cells. Healthy. Left side shows very much contracted mastoid of the diploëic type, with evidence of disease located at the knee of the sinus and extending down to the antrum. The lateral sinus is clearly outlined, while in the right side it can scarcely be recognized. (Diseased area marked with arrows.)

Operative Findings. 1. Periosteum thickened and adherent.

2. Cortex thin.

3. Fistula present opposite the spine of Henle and cavity underlying it.

4. Pus thick but not under pressure

5. The rest of the cells were filled with pus, granulation tissue, and most of the intercellular septa were still present.

6. The mastoid cavity was large and deep and I was unable to locate the antrum, so I closed the wound completely.

Laboratory Report. Pus returned sterile, no growth on blood agar and Löeffler's blood serum.

CASE XI.—W. F., aged one and a half years. (January 12, 1923.)

History. Six months previous to this ailment had acute suppurative otitis media with spontaneous cure. On January 23, when the baby was first seen by me, I noted an acute suppurative otitis media with a postauricular edema. This was secondary to measles. Incision revealed a postauricular abscess secondary to scalp infection.

X-ray Report. Mastoids poorly developed. The right mastoid shows a few cells. The cell walls are not clearly defined. The left mastoid shows no evidence of cells and is apparently of the diploëic type, or possibly sclerotic. Signs of disease indefinite. (Diseased area marked with arrows.)

Conclusion. The postauricular abscess produced a shadow and the previous suppurative otitis media caused a slight sclerosis, which in this instance could have been misinterpreted for some serious pathology.

CASE XII.—E. S., aged nine years. (December 22, 1922.)

History. This patient had an acute suppurative otitis media, complicated by rhinitis, with spontaneous rupture of the ear-drum. When first seen she had profuse discharge and severe pain in the ear.

Examination. Impaired hearing. Loss of whispered sound. Spoken voice audible at 3 feet.

Laboratory Findings. Leukocyte count, 9000. Urine negative.

X-ray Report. Left mastoid well developed, pneumatic in type, large cells. Area extensive. Right side shows mastoid pneumatic in type. Cell walls indistinct and at places entirely destroyed. (Diseased area marked with arrows.)

Operative Findings. 1. Periosteum thick and adherent.

2. Cortex thick.

3. Complete liquefaction of the mastoid cells with a perisinus abscess.

4. Cavity large and antrum very deep.

Case XIII.—J. C., aged sixty years. (March 18, 1922.)

History. Patient has been ailing and losing weight for the past three years, suffering with diabetes and boils all over the body. He developed pain in the right ear and he called in his son, who is a physician. It was thought at that time that he had furunculosis of the external auditory canal. After several days of treatment, he consulted a neighboring otologist, who incised the edema in the canal and suppuration followed. His symptoms were not alleviated, and after thirteen days I was asked to see him.

Examination. Profuse aural discharge. Eczema of the external auditory canal and auricle. The pus was pulsating. The ear-drum was almost completely destroyed. His hearing was impaired and the temperature was never above 101°. The most annoying symptoms were severe headache and hemicrania, which were worse at night. He was treated expectantly for three weeks, after which he was sent into the hospital for observation. Pain over mastoid on deep pressure only.

Hearing Tests. Loss of low tones. Weber lateralized to the right. Reinné negative. Bone conduction diminished.

Laboratory Findings. Urine, 4 per cent sugar present. Albumin present. A few hyaline casts present.

X-ray Report. Mastoid of the pneumatic type. Small cells. Medium-size area on the left. On the right the area corresponds to the left. The cell walls are destroyed. The wall of the sinus is indistinct. There is destruction in the region of the antrum and increased transparency above this area. The oblique view shows thickening of the cortex. (Diseased area marked with arrows.)

Operation. Local anesthesia, 1 per cent novocaine. Complications. Infection of the neck.

Operative Findings. 1. Thickened periosteum and very adherent.

2. Thick cortex.

3. Large quantity of pus, and pulsating.

4. Perisinus abscess. The whole sinus was exposed and covered with granulation tissue.

5. Extradural abscess with a complete loss of the dural plate from the antrum all along the roof of the mastoid cavity.

6. Many cells filled with necrotic mucous membrane, posterior to the lateral sinus. When found the mucous membrane was edematous and of a dark bluish color and very thick.

Case XIV.—M. L., aged forty-eight years. (December 13, 1923.)

History. Patient had rhinitis complicated by an acute suppurative otitis media. There was a profuse discharge with severe mastoid tenderness and a slight toxic facial palsy. On December 13, 1921, she went to the hospital for observation. After being there for one week her condition cleared up. This again emphasizes the point that large cells may empty themselves. The patient returned to me May 11, 1922, with an acute exasperation of these symptoms. The ear-drum was again incised and *x*-rays taken. Operation was suggested, but the patient refused. She now has a *chronic* suppurative otitis media.

X-ray Report. Mastoid of the pneumatic type, large cells, extensive in area, clearly distributed anteriorly and posteriorly to the lateral sinus. On the left side the cells are filled with exudate, but the cell walls are still visible. (Diseased area marked with arrows.)

Case XV.—H. B., aged two years. (March 13, 1923.)

History. Patient had measles complicated by an acute suppurative otitis media, bilateral.

Examination revealed profuse aural discharge. Smoothing of postauricular sulci. Extreme tenderness over mastoid on palpation. Displacement of right auricle.

Laboratory Findings. Leukocyte, 26,000. Urine negative.

X-ray Report. Mastoid cells unusually well developed for this age. Both sides show disease. On the right the disease is present in the neighborhood of the knee, indicated by a general clouding. On the left the disease extends out into the zygomatic area with destruction of the cell walls (Diseased area marked with arrows.)

Operative Findings. 1. Right side: Periosteum thin, stripped very easily.

2. Perforation of cortex on the right side with extensive necrosis over the zygomatic region.

3. Left side: There was liquefaction of the zygomatic cells, and there seemed to be a distinct bony shell dividing the zygomatic cells from the rest of the mastoid cells.

Case XVI.—B. F., aged five years. (March 15, 1923.)

History. Patient had measles complicated by otitis media, bilateral. There was spontaneous rupture of both ear-drums. When seen there was a profuse aural discharge with septic temperature and pain over both mastoids. Both ear-drums were incised three times. This condition continued without improvement. There was a progressive loss of hearing, and with the profuse discharge I felt that an operation was imperative.

Laboratory Findings. Leukocyte count, 15,900. Urine analysis negative.

X-ray Report. Mastoids on both sides extensive in area. Both sides show disease. Examination made March 15, 1923, showing a general clouding of the cells on both sides. Cell walls still visible. On the left side there is a definite localized absorption just below the knee of the sinus and extending over the sinus. The cell walls show some absorption on the right side near the apex, March 19, 1923. The evidence of disease and lime absorption has increased on both sides at the area previously described. (Diseased area marked with arrows.)

Operative Findings. 1. Periosteum thickened but not adherent.

2. Large cavity at the tip on the right side with complete breaking down of the zygomatic cells. Left side: Complete breaking down of the zygomatic cells with extensive necrosis of the cells behind the lateral sinus.

Conclusion. The mastoids were *x*-rayed, March 15 and March 19, 1923 (four days apart). The comparative study showed that there was a progressive absorption taking place during this interval. The operation was performed on the 22d of that month (a three-day interval). Our operative findings here showed more pathology than the *x*-ray, but I feel that we are not to condemn the *x*-ray, as there was a three-day interval, and so much more destruction took place.

CASE XVII.—J. S., aged thirty-six years. (October 22, 1923.)

History. Patient began with an acute rhinitis, and after fourteen days developed pain in the right ear. Four days later he came to my office suffering with pain in the ear and pain over the right side of the head. Examination revealed a bulging ear drum which was incised under ethyl-chloride anesthesia. (This was on October 22, 1923.)

Previous History. Patient gives a history of having had scarlet fever during childhood and also a discharging ear. On November 23, 1923, he returned, complaining of severe pain over the mastoid. He had a profuse aural discharge and a marked loss of hearing.

X-ray Report. Mastoids of the pneumatic type. Symmetrical. On the right side there is evidence of disease, showing at the tip and involving the area posterior to the lateral sinus. There also appears to be disease in the region of the antrum. (Diseased area marked with arrows.)

Operative Findings. 1. Periosteum thick and adherent.

2. Cortex very thick and sclerosed.

3. After deep chiseling, we found an area of necrosis at Macewen's triangle.

4. Liquefaction of the deep cells in the zygomatic region.

5. Cavity at the tip and a large cell filled with pus posterior to the lateral sinus.

6. Lateral sinus exposed and covered with granulation tissue. Temperature normal.

Conclusion. Here we have a history of inflammation and discharging ears during childhood which probably explains the

sclerosis of the mastoid bone. Only the deeper layer of the cells were involved, as there were no superficial cells. Clinically the tender points corresponded to the *x*-ray and operative findings. These tender points were at the tip, over the posterior edge of the mastoid and over the zygomatic area.

Case XVIII.—F. C., aged five years. (May 26, 1923.)

History. Patient had grip and rhinitis complicated by acute suppurative otitis media. Both ear-drums were incised four times without abatement of the symptoms. Child was toxic and very tender over both mastoids. He had profuse aural discharge and pain over tip on the right side and pain over emissary on the left side.

X-ray Report. Mastoids of the small-cell pneumatic type. Rather extensive in area, considering the age of this child. A definite clouding of the cells on the right side. Cell walls still visible. Left side shows destruction of the cell walls about the tip. (Diseased area marked with arrows.)

Operative Findings. (Bilateral operation.) 1. Right side: Periosteum not thickened and not adherent.

2. Cortex medium.

3. Cavity at the tip.

4. The rest of the mastoid cells were soft, filled with pus, but not liquefied.

5. Left side: Nothing unusual about periosteum on this side.

6. Cortex congested and of medium thickness.

7. Cavity opposite spine of Henle.

8. Small cavity at the tip.

9. Zygomatic cells necrotic.

Conclusion. In bilateral suppuration it is natural to operate on the side that shows most symptoms and most destruction on *x*-ray plates. During this operation I associated the shadows seen on both plates, and since both mastoids are usually alike, I felt that I would find sufficient pathology to warrant mastoidectomy.

CASE XIX.—I. S., aged seventy-two years. (January 25, 1923.)

History. Patient was ill with pneumonia for three weeks and suffered with pain in the ear for one week. Ear was incised by another otologist and pus was found.

Examination. 1. Profuse aural discharge.

2. Pain all over mastoid.

3. Thickening of the periosteum over mastoid.

4. Nipple-shaped ear-drum.

X-ray Report. Mastoid of the pneumatic type, extensive in area. Symmetrical. General area clouded by exudate. There is absorption of lime salts in the neighborhood of the lateral sinus and extending posteriorly. (Diseased area marked with arrows.)

Operative Findings. 1. Periosteum thick and stripped with difficulty.

2. Cortex thick.

3. Pus under pressure.

4. Mucous membrane very thick, and bleeding from the bone was very profuse.

5. Sinus superficial when broken into.

6. Large cavity posterior to the lateral sinus.

7. Extensive necrosis of the posterior canal wall.

Operative. Local anesthesia, 1 per cent novocaine.

CONCLUSION. 1. *X*-ray is indicated as a routine whether the diagnosis has been made or not, so as to correlate clinical and operative findings.

2. It is important to cleans the area to be *x*-rayed, as ointments used as medicaments may contain some metallic substances which will produce pseudoshadows.

3. Some atypical cases probably would never be diagnosed without an *x*-ray (as in Case VI–R.), where we had no middle-ear suppuration, and at operation we found an extradural abscess.

4. *X*-ray, in my experience, has always been very helpful, and at no time was I inclined to operate unless the clinical symptoms warranted such procedure.

5. The mastoid pathology can be watched as to progress (as was shown in Case XVI), by means of *x*-ray at three-day intervals.

6. Those of us following the conservative treatment in mastoid therapy will bear in mind that it is important to keep vigilant watch on the lateral sinus. Should there be any signs of haziness or complete loss of the lateral sinus from view, we may suspect necrosis of the anterior bony wall with a perisinus abscess.

7. It is especially important for the novice beginning to practise in our specialty to take routine *x*-rays, as he may learn the topographical and regional anatomy of the mastoid, as to type, whether cellular or non-cellular and position of the lateral sinus.

8. The angle from which the mastoid picture is taken must be understood, as otherwise the clinician will misinterpret some of the shadows.

9. Cavity formation appears as a blurred area without septa and the edges of the cavity are not distinctly visible. This must be differentiated from a large normal cell which is irregular in shape, the edges of which are distinct, sharply outlined with septa intervening.

10. In order to obtain good pictures in children, I ask to administer a few whiffs of chloroform when necessary.

11. In children where cells are not fully developed, I always study the plates along the posterior canal wall, as in this area the bone is thickened and the antrum has one or two cells, and if there is any disease it should be seen there. (This condition was especially true in Case IX.) In this case the semicircular canals were distinctly visible, and the only pathology noted was around the antra.

12. In mastoid disease always *x*-ray both mastoids and compare the normal side with the diseased. In most instances the cellular distributions and general confirmation of the mastoids are alike, and any deviation from the normal will readily be seen unless there is a history of previous middle-ear suppuration.

13. In bilateral suppuration both plates should be studied, selecting the side to operate which shows most pathology from a clinical and radiographic standpoint.

14. The operative findings in one mastoid will usually interpret the plates of the opposite side and a decision as to the course of pursuit will be simpler.

15. It is the idea of many clinicians to *x*-ray all mastoids so as to correlate the *x*-ray with the clinical and operative findings. Although we may make our diagnosis in most cases clinically and judge whether operation is indicated or not, it should, however, be our aim to develop clinically so as to be able to outline the pathological conditions underlying the mastoid cortex.

16. Repeated *x*-rays will show whether the disease is at a standstill, progressing or undergoing sclerosis. (Case XVI–F. showed progressive destruction while Case III–T. showed the condition at a standstill.)

17. We must bear in mind that operations are often delayed after *x*-ray plates have been taken, and radiographic studies must therefore not be condemned because they do not coincide with the operative findings.

I desire to cordially express my sense of indebtedness and gratitude to my friend and able chief, Dr. George M. Coates, to Dr. Benjamin Shuster for his valuable assistance and to Dr. George P. Pfahler for his able and conscientious aid and collaboration.

NOVEMBER 21

Studies of Mastoid Disease by the *X*-rays, with Operative Findings. Demonstration of a Special Localizer[1]

BY G. E. PFAHLER, M.D.

In most large otological clinics the examination of the mastoid by means of the *x*-rays has become almost a routine procedure. The value placed upon these studies in each clinic is generally

1 Presented with lantern-slide demonstration in conjunction with the clinical observations and operative findings, by Matthew S. Ersner.

measured by the technical skill which has been developed in making roentgenograms of good quality, but more particularly by the intelligent and skilful interpretation. The clinicians who have had the most experience with this aid in diagnosis are the ones who are most enthusiastic. I know of no roentgenologist who will ask a clinician to depend entirely upon the roentgenographic evidence in mastoid disease, especially in deciding upon operation.

The *x*-ray picture of the mastoid is often interpreted with difficulty and requires keen observation, a thorough knowledge of the conditions which cause variations in density and a consideration of the technic with which the film was made. There is almost as much variation in the type and distribution of the mastoid cells as there are variations in the type of noses on peoples' faces. For this reason both mastoids are always studied. Generally they are symmetrical, but Dixon[1] in a study of 522 cases found asymmetrical mastoids in 14.5 per cent. Auberg[2] in a study of 120 pairs of mastoids found 82 symmetrical and 28 asymmetrical.

Law[3] divides the mastoids for roentgenographic studies into the "non-cellular" and the "pneumatic or cellular." The "non-cellular" consist of three types, the undeveloped, or infantile, the diploëtic and the sclerotic. The pneumatic, or cellular, consist of the large cell, the small cell and the mixed cell. These two general types may be mixed, and rarely one may have a pneumatic type on one side and a diploëtic type on the opposite side. Cheatle[4] calls especial attention to the infantile type, which existed in 20 per cent of all the cases he examined and persisted into adult life in 1 or 2 per cent. The common characteristic of these infantile types is the dense outer antral wall. The lateral sinus is generally much farther forward than in the cellular types. Gerber[5] cautions against interpreting these infantile types as "sclerotic," and says that in the infantile type a chronic infection can be prevented by early drainage of the antrum, regardless of the absence of the classical mastoid signs.

This variation makes interpretation difficult. Spencer,[6] in a review of the *x*-ray reports of 100 cases of mastoiditis, at Base Hospital, Camp Lewis, Washington, found the *x*-ray reports

confirmed at operation in 80 per cent. The 20 per cent included all doubtful cases and all cases in which the *x*-ray report, or the operative report, was very briefly or incompletely written. He believes that if both reports had been completely written there would have been confirmation in a much larger percentage.

In the series of cases studied by Dr. Ersner and myself the rays failed to show disease in a child of sixteen months and in a man of seventy-three years. In the case of the old man the disease was not recognized in the *x*-ray film, but a review of the films shows the evidence of the disease, as was found at operation. In the case of the young child the failure to show disease was due to lack of development of the mastoid cells. In the case of the child even the semicircular canals were shown yet no evidence of disease could be recognized. Law says, "Except in very unusual cases the mastoid process does not show cellular development before three years of age, and usually not until about the fifth year of age." We are glad to say that in a number of cases of children we have been able to recognize disease under three years of age.

A satisfactory roentgenogram should show the external auditory meatus, the internal auditory meatus, the mastoid antrum, the temporomaxillary joint, usually the outline of the lateral sinus, as well as the middle and posterior cranial fossæ. Frequently the ossicles of the ear are shown and at times in children the semicircular canals. One should always keep in mind the type of mastoid, and this should be described definitely.

Technic. The first essential in the roentgenographic examination of the mastoid is the making of good films, which will show the finest possible details. The second essential is skilful interpretation and the third essential is the correlation of the *x*-ray evidences with the clinical symptoms. In so far as any of these three are lacking there will be failures.

1. The patient's head must be absolutely fixed. We accomplish this with sandbags.

2. The central ray should be passed directly through the auditory canal. This I have been able to accomplish by means of a special localizer which I am demonstrating to you and which will

be described in detail in a subsequent paper. Briefly, it consists of a head rest which permits us to absolutely fix the external and auditory meatus in direct relation to the tube-holder, so that the two sides of the head can be examined exactly the same, and any two heads can be examined alike. The central ray is passed obliquely from above at an angle of 10 degrees and posteriorly at an angle of 15 degrees. This makes the central ray enter the proximal side of the head about $1\frac{1}{2}$ inches above the external auditory meatus about $1\frac{3}{4}$ inches posteriorly to the external auditory meatus. The films may be made stereoscopically, as is recommended by Allen[7] and others. We do not make stereoroentgenograms routinely, because this of necessity changes the direction of the central ray. In certain special cases stereoroentgenograms are of advantage.

3. Sharpness in detail is improved by the use of a small cylinder diaphragm. We use one cylinder, 3 inches in diameter, at the surface of the head and then make a second film on each side with a cylinder $1\frac{3}{4}$ inches in diameter. Such small diaphragm work is only possible where an exact localizer is used.

4. The duration of the exposure will vary with the individual technic. We use superspeed films, double screens, a $3\frac{1}{2}$ or 4 inch parallel spark gap and 4 to 6 seconds' exposure.

In addition to the positions described above, one may often get special information with regard to the tip of the mastoid by directing the central ray through the mouth toward the tip on the opposite side. In whatever position, both mastoids should be examined and compared.

Hickey[8] has described an anteroposterior position for the mastoid. Each side is exposed separately with the patient lying on his back. The tube is tilted 5 degrees. The central ray is passed through the tip and a short exposure is made.

Sometimes additional information may be obtained by oblique exposures, either projecting the shadow of the skull forward, by directing the central ray from behind and from below, or one may project the cells into the occipital region by directing the central ray from above and anteriorly to the petrous portion of the tem-

poral bone on the proximal side. This position will project the dense petrous portion below the level of the antrum.

Gerber has described, but not yet published, what he calls the "occipital projection," which is obtained by introducing the central ray nearer to the occiput and obtaining more detail about the antrum.

INTERPRETATION. It is usually necessary that the clinician correlate the clinical symptoms with the interpretation by the roentgenologist. However, it is not always necessary that the clinical symptoms correspond to the *x*-ray findings. In some cases the local clinical symptoms may be absent. Lange says, "The mastoid may be the seat of an infection of either recent or remote origin, unheralded by a single symptom," and that no less an authority than Politzer made the statement that the most extensive necrosis may occur in the mastoid without giving rise to a single symptom.

Since about 85 per cent of the mastoids are symmetrical, one can generally make a direct detailed comparison of the two sides. When both sides are diseased, or when there is asymmetry, the roentgenologist must use his general knowledge of diseased appearances as compared with the normal in the same mastoid as well as in other patients. The normal mastoid of whatever type gives the appearance of a network of continuous cancellous lines, corresponding in density to the surrounding healthy bone. The general evidence of disease is indicated by exudate in the cells, by destruction of the cell walls or by evidence of repair, as is shown by sclerosis. It is necessary for the surgeon to know the type of mastoid, the degree of involvement and the presence or absence of bone destruction.

Acute Mastoiditis. In the minor grade there is a cloudiness of the cells, with the cell walls or trabeculæ still clearly visible. One must be quite sure that the cloudiness is not due to a faulty or fogged film. The cloudiness may also be due to swelling in the soft tissues, as from furunculosis. The sinus groove stands out more clearly, and in early acute mastoiditis the region of the antrum is relatively more opaque than the body of the antrum.

It must be borne in mind that this cloudiness may be due to pus or to thickened mucous membrane or granulations.

Severe Acute Purulent Mastoiditis. The pneumatic type show a marked degree of opacity over the entire mastoid area. The cell walls may be completely obliterated, and one detects evidence of absorption of lime salts. This indicates severe active disease, but not always necrosis or destruction of the cell walls. If the absorption of lime salts is localized or if the cell walls are totally destroyed it is likely to indicate necrosis. The disease is most likely to be shown in the antrum or at the knee of the sinus. In the stage of lime absorption the anterior wall of the lateral sinus is apt to become less distinct, because of absorptions of lime salts.

Mastoid Abscess. Abscess is indicated by a destruction of the cell walls, which may involve the entire mastoid. If it occurs about the wall of the lateral sinus it becomes a perisinus abscess. It may break through the cortex and give local external symptoms, or it may break through the tegmen tympani and become an epidural abscess. The abscess is indicated by an area of increased transparency as compared with the surrounding tissues.

Chronic Mastoiditis. This condition is indicated by a general sclerosis very similar to an undeveloped mastoid. A close search should be made for any remaining cells or any evidence of bone necrosis or sequestrum. The history of a persistent or chronic recurring purulent discharge will serve to differentiate this condition from the undeveloped mastoid.

Cholesteatoma. The process is inflammatory and the edges of the cavity show an osteitic change, a thickened border indicated by increased density. Law says "Cholesteatoma always occurs in a chronic process or in an undeveloped or sclerotic type of mastoid." The discharge is usually foul and will show the presence of cholesterin crystals.

In this clinical study of 142 cases, in association with Dr. Ersner, many of the films were made at the Medico-Chirurgical Hospital and interpreted by my associate, Dr. B. P. Widmann. In only two instances did the operator fail to confirm the *x*-ray findings. In one of these the child was only three years of age and the mas-

toid cells were undeveloped. In the other case the disease was shown in the films and can be seen now, but was not properly interpreted. This simply indicates that skill is required in interpretation and is no reflection on the method. In all instances the disease was suspected by Dr. Ersner from the clinical symptoms. This means a confirmation in about 98 per cent.

In a series of cases studied at the Polyclinic Hospital, where most of the films were made and interpreted by my assistant, Dr. William Campbell, with review by myself, Dr. Gleason tells me that in practically all cases the diagnosis was confirmed at operation.

Conclusions. 1. The *x*-ray examination should be a part of the routine study of each case of mastoid disease.

2. In all instances the evidence should be correlated with the clinical evidence of disease, especially in deciding upon operation.

3. Success depends first upon skilful technic in making fine roentgenograms, and, secondly, in careful observation and interpretation.

4. The two sides should always be examined and generally compared, but they are not always symmetrical, and interpretation must often be made independently of the opposite side.

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The Radical Frontal Sinus Operation, with Results in Sixty-five Cases

BY FIELDING O. LEWIS, M.D.

The treatment of chronic suppuration in the frontal sinuses, in the vast majority of cases, calls for some form of radical surgical procedure. The external route offers the only means by which the frontal sinuses can be properly inspected, all disease removed and adequate nasal drainage instituted. These are the essential factors in procuring complete permanent cessation of all purulent discharge, together with the thorough removal of the ethmoidal sinuses, when necessary.

In this connection it is interesting to note the development of the present-day external frontal sinus operation.

Before Ogston's paper on "Trephining the Frontal Sinus," which appeared in the *Medical Chronicle*, 1884, the method of treating frontal-sinus suppuration was by external drainage through a trephine opening in the anterior plate, a method, I am sorry to say, we sometimes find in use today. Ogston was the first to call attention to the importance of free nasal drainage, and it was he who first described enlarging the nasofrontal duct, followed by introducing a drainage tube into the nose from the frontal sinus. Ten years later the skin incision and method of drainage were slightly modified by Luc; since that time the operation has been generally known as the "Ogston-Luc" operation. Jansen, in 1894, described his operation in which the entire inferior wall was removed, mucous membrane scraped away and free nasal communication established. Kuhnt, a year later, advocated the obliteration of the sinus by the removal of the entire anterior wall and nasal portion of the floor of the sinus, curetting the infundibulum and anterior ethmoidal cells. Reidel, in 1898, was apparently dissatisfied with the results he obtained from the above-mentioned operations, so he operated by removing entirely both the anterior and inferior walls, thereby obliterating the sinus by allowing the soft parts to adhere to the posterior wall. It does not require a keen observer to see why this method was not universally adopted, especially in large sinuses. However, I have seen cases in which this type of operation was necessary on account of the extensive necrosis.

It remained for Killian, in 1902, to describe his classical operation for obliteration of the frontal sinus, with a minimum amount of deformity, by preserving the well-known Killian bridge. This operation, or modification of it, is the one generally in use by the surgeons of today. In the literature on this subject, during the last four or five years, one can see a trend toward less radicalism, especially to be noted in this country. I am of the opinion, however, that there is a certain group of cases in which the Killian operation offers the best means of obtaining a cure.

The chief indications which are given for the radical operation are: (1) failure of the intranasal operation; (2) presence of fistula, abscess or indications of necrosis; (3) symptoms of intracranial complications; (4) pain and fever with foul discharge from the frontal sinuses; (5) persistent headaches associated with ocular discomfort and not relieved by intranasal treatment; (6) the presence of new growths within the sinuses.

The Killian operation is so well known that its description at this time is unnecessary. I wish, however, to mention a few minor modifications which I have found useful in my cases: (1) suturing the eyelids together is a great help in protecting the eye from injury; (2) infiltrating the skin and deeper structures with a one-half of one per cent novocain and 1 to 10,000 adrenalin solution. By this means hemorrhage is very much reduced. This method has recently been mentioned by a Viennese surgeon, and is the method also used when the operation is to be performed under local anesthesia. In addition, I prefer to use general anesthesia by the intratracheal method. (3) The incision as described by Coakley, namely, instead of one curved incision extending through the eyebrow around the side of the nose and ending below the eye, two incisions are made. The first, or upper incision, is made through the upper margin of the eyebrow and extending through the periosteum to the bone. The lower incision is started posteriorly to the supraorbital notch and carried in a curved direction downward along the side of the nose, midway between the inner canthus of the eye and dorsum of the nose, to the inferior border of the nasal bone. This prevents puckering at the inner canthus of the eye, which is so frequently seen after the single Killian incision. Imperceptible scars are frequently obtained in this

manner. Here, also, if one is careful that the external skin scar is not directly over the resected bone, a depressed scar will be obviated. (4) In extremely large sinuses, which are divided by septa, no overhang is left in the upper margin, the septa are partially removed and thoroughly examined for accessory cells. In so doing every crevice is explored and all mucous membrane removed. In my experience I have frequently found diseased cells in the body of the septa, which, when not removed, have resulted in failure.

The osteoplastic operation I have never used because the principles never appealed to me.

Walter G. Howarth, of Edinburgh, described in the July number of the *Journal of Laryngology and Otology* of this year an operation which to me seemed quite adaptable to certain cases. I have followed his method in my last 4 cases with very gratifying results. This operation, I feel, merits a brief description for the benefit of those who have not seen or used it. He calls attention to the objection brought forward to the two operations commonly in use, namely, the Killian operation and the Ogston-Luc operation. In the Killian operation, he points out, dead spaces are apt to occur behind the bridge, along the ethmoid and elsewhere, which give rise to further trouble. The Ogston-Luc operation, while improving the drainage of the sinus, cannot ensure a thorough removal of the ethmoid.

The following is the author's technic: A curved incision is made just under the supraorbital margin and brought down in front of the inner canthus on to the side of the nose. The skin being very thin in this region, a fine scar is obtained. Moreover, as it is in this situation that the shadow falls, often the scar is almost invisible. The incision is carried down to the bone; the periosteum covering the roof of the inner wall of the orbit is then raised; the pulley of the superior oblique is thus detached from its notch and the whole of the orbital contents displaced outward; likewise, the duct is displaced from its groove and retracted outward with the orbital contents.

The sinus is now opened in the situation where one is always likely to find it, just above the lacrimal groove, and with Citelli forceps the whole of the floor of the sinus is removed right up to

the supraorbital margin. The lining mucosa should be disturbed as little as possible; that which lines the dome of the cavity should be left intact. With Citelli forceps the bone in front of the frontonasal duct (that is to say, part of the ascending process of the superior maxilla) is removed. The ethmoid is next attacked, entrance being effected through the lacrimal groove. The ethmoid cells are removed backward, if necessary, to the sphenoid. A new frontonasal duct is then formed farther forward than the old one. In order to effect this an additional portion of the ascending process of the superior maxilla and of the nasal process of the frontal bone is removed with gouge and forceps. A large, firm-walled, rubber drainage tube pushed up the nose will lie well forward, the upper end being tucked into the anterior part of the sinus and the lower end stitched to the ala of the nose. The orbital contents are then allowed to fall back into place and the incision completely closed with fine silkworm gut. The tube is removed in ten days.

I have operated radically on sixty-five frontal sinuses; included in these are the cases on whom more than one operation was performed on the same sinus. Fourteen cases had secondary operations. The failure of the first operation I found to be due to a lack of nasal drainage, or unnoticed accessory cells, which had not been carefully treated.

There were 4 deaths; 2 from multiple brain abscess and 2 from meningitis, confirmed by autopsy. One of the cases of brain abscess and 1 of the cases of meningitis were in precarious condition at the time of operation. One case of the series required removal of an eye as a result of a secondary infection following a sinus operation. Ten of the cases were operated on bilaterally. All but 9 had involvement of ethmoids. Thirteen had antral disease, requiring radical operation. Three had previously been operated on by the intranasal route and 5 had radical operations before coming under my care. In 1 a foreign body was the cause of continued infection, in another multiple bone cysts were encountered, the cysts containing a straw-colored fluid. The first case in the series developed a mucocele three years after the radical operation. Three cases had exposure of the dura from necrosis of the posterior plate. There were no intracranial com-

plications following the operation. Three cases were sufferers from asthma, 2 of which were relieved by operation, the remaining 1 greatly improved. In 3 cases the disease was the direct result of an injury, 1 caused by the kick of a mule, another from a flying piece from a gasoline motor and another from a direct blow over the sinus. Three of the cases, with bilateral disease, were operated through one side, and all made a very prompt and uninterrupted recovery. When last seen all of the living cases were entirely free from discharge.

Operations and cases were demonstrated by lantern slides.

DECEMBER 19

The Anatomic and Physiologic Importance of the Endolymphatic Organ

BY PROF. GEORGES PORTMANN
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(Translated by Dr. Leon Felderman, Philadelphia)

The internal ear, unfortunately, is still imperfectly known, and this helps to explain why its pathology is always so confusing. For a long time I have been struck by this fact, that the authors who have been studying the labyrinth have not sufficiently interested themselves in the endolymphatic sac. Even Retzius, whose work on the internal ear is very considerable, has not considered the endolymphatic sac of great importance. This is surprising to note, when we consider its dimensions and the bearing it has on surrounding structure, such as the cerebellum and the auricular cavities, situated in the intrapetrous portion of the temporal bone. In order to understand the human anatomy it is indispensable to study the comparative anatomy. It is with this aim that I have undertaken, for the past few years, to study the endolymphatic sac in all groups of the vertebrates: Selachians, teleosteans, batrachians, birds and mammals. In all these groups I have noticed the presence of the endolymphatic organ, its considerable dimensions, and its relation to the ocean in the case of certain fish, or to the meninges in the case of mammals.

In the case of human beings, I have studied the bony bed in which the endolymphatic organ is situated. It is here that I have been influenced to describe a fossa on the posterior surface of the petrous portion of the temporal bone, which I term the *endolymphatic fossa*. It is of triangular form, with its peak forming a right angle; it extends on a large surface just to reach the lateral sinus. The bottom of the small fossa is smooth, while the posterior surface of the petrous portion is always rugged. On the level of its superior angle opens the aqueduct of the vestibule canal, situated in the interior of the petrous portion of the temporal bone and placed in communication with the endolymphatic fossa and the intrapetrous vestibule.

But this study of the bony labyrinth is the introduction to the study of the membranous labyrinth. In the endolymphatic fossa one finds the endolymphatic sac. This sac rests on the posterior surface of the petrous bone in the fossa and adapts itself to its shape. It is completely included in the dura mater and terminates in the lateral sinus. The cerebellum and its arachnoid spaces adapt themselves, so to speak, to the posterior surface of the petrous bone, and in this way act as a veritable physiologic drum, receiving all the variations of pressure, be it of the cerebellum, the arachnoid spaces or the intracranial circulation of the lateral sinus and its intermediary. It is easy to recall that the intracranial cavity, wherein is situated the cerebellum, is almost completely closed above by the tentorium cerebelli, and consequently any modification of pressure will register in a more sensitive manner.

The endolymphatic sac continues by way of the endolymphatic canal, which penetrates into the aqueduct of the vestibule and arrives in the interior of the petrous bone in the vestibule cavity. There the endolymphatic canal dilates to form the saccule. This saccule communicates with the cochlear canal in one part and the utricle and the semicircular canals in another. We have therefore a double-sacced organ of which the two extremities are dilated, one in the interior of the petrous bone, the saccule, the other in the cerebellar cavity, the *endolymphatic sac*. All intracranial pressure modifications are therefore directly transmitted to the internal ear.

In the study of the anatomy we have shown the importance of the endolymphatic organ, and it is interesting to study its physiological value. It was important to find among the animals some in which the endolymphatic organ was superficial, and one could destroy this organ without disturbing the internal ear. Following the principle of the great physiologist, Claude Bernard, one should examine the disturbance following functional suppression of the endolymphatic organ, deducting its physiologic value. The species of fish selachians, owing to their anatomic peculiarity, are the most favorable for scientific experiment. In fact, in these animals of the ocean, the endolymphatic organ opens in the posterior region of the head by a small canal. It was consequently close to the canal opening. I do not wish to describe the detail of the technic. Suffice it to say that the most approved method is by employing the thermocautery with a very fine point. A light cauterization will form an epithelial cork and obliterate the endolymphatic canal.

The animals operated upon were the Leiobatis Pastinaca of the Ragides family, which later had their heads cut for histologic examination. It was necessary first that their internal ears be intact; second, that the endolymphatic canal be well obliterated.

The result of my experiments has passed expectation, and in spite of difficulties presented by moving pictures under the water, I successfully filmed the disturbance presented by the animals operated, all of which had disturbance of equilibrium and vertigo shown in the different angles of swimming, they being unable to hold the horizontal position, and falling more vertically or leaving the water in turning from side to side. At times the disturbance of equilibrium was both vertical and horizontal and would make them descend in spiral form. In these experiments and in those which I actually followed in a mammal, I expect to be able to prove the importance of the endolymphatic organ as a regulator of endo-auricular pressure and it is easy to understand, from a pathologic standpoint, why modification of the endocranial pressure could provoke, through the intermediary of the endolymphatic sac, a functional disturbance of the internal ear.

This is one new route for research which could perhaps help us in clearing the physio-pathologic condition of the labyrinth.

PROCEEDINGS

OF THE

SECTION ON GENERAL MEDICINE

JANUARY 22, 1923

Alcoholic Injection of the Gasserian Ganglion for Tic Douloureux

BY GEORGE N. DORRANCE, M.D.

AND

J. WILLIAM BRANSFIELD, M.D.

PHILADELPHIA

The use of alcoholic injections into the various divisions of the fifth nerve outside of the skull for the relief of trigeminal neuralgia is an established procedure.

The relief obtained varies from one to eighteen months, dependent upon the infiltration.

It is the injection of the ganglion itself that we are interested in, and here a wide divergence of opinion exists both as to the rationale of the method and regarding the length of time during which relief is obtained. We are inclined to believe that in the hands of an experienced operator, when a successful injection into the ganglion is made, the relief is permanent.

In 1911 Taptas, of Constantinople, made the first successful subcutaneous injection of alcohol into the ganglion, while attempting to inject the third division, although Sicard, in 1910, injected the ganglion after exposing the foramen.

In 1916, we published an article on "The Technic of Injecting the Gasserian Ganglion with Alcohol,"[1] and since that time we have been employing this method. We are still of the opinion expressed at that time that the operation advocated by Spiller and so successfully performed by Frazier, is the ideal method in the hands of experts where a major operation is desired by the patient.

We have had cases referred to us where an "open operation," was absolutely refused both by the patient and the physician, and in these cases we have injected the ganglion with perfectly satisfactory results.

Our earliest case is one of seven years' standing, with complete relief of all pain and complete anesthesia in the area supplied by the fifth nerve.

The objections raised to the use of alcoholic injections into the ganglion have been based on the earliest cases done when the technic was not perfected and when operators with little skill or knowledge of the principles involved were using what was believed to be a simple procedure.

The same training and skill are required to inject the ganglion with alcohol as to remove it. No one without practising faithfully on cadavers for a long period of time and without a thorough knowledge of the deep anatomy of the parts should attempt the injection.

Dr. Harvey Cushing is especially bitter in decrying alcoholic injections into the ganglion. He has never deliberately injected the ganglion but records cases he has seen where failures of various kinds have occurred. He cites the danger of the lower jaw becoming locked from alcohol getting into the joint; middle-ear diseases with sloughing of the parts due to alcohol being injected into the Eustachean tube; facial paralysis, etc. In practically all of the cases cited by Cushing, an error in technic occurred, and like any other operation unsuccessfully performed, unfavorable sequelæ ensued.

[1] Pennsylvania Medical Journal, August, 1916.

We do not claim that alcoholic injection into the ganglion is free of all complications. None of a serious nature, however, has occurred in our cases. In no case was the patient dissatisfied with the minor complications which were more than compensated for by the complete absence of their pain.

The opinion of medical men in Philadelphia, judging from the statements made by physicians referring cases to us and our hospital interns, is that the relief of pain from ganglionic injection is very temporary, varying from nine months to one year. We wish to correct this erroneous impression. We have never had a recurrence of any of our cases where the ganglion was successfully injected.

Hartel, in 1920, reports 50 cases with no recurrences. His earliest case at that time was of seven years' duration. Byrnes, in the same year, reports 4 recurrences in a series of 30 cases. His earliest case at that time was of six years' standing.

In preparing this paper we wrote to all of our cases, but only 8 have replied to date. Three of these cases were of more than four years' standing; 1 was six and one-half years; another was seven years, and all were free from pain. The remaining 3 cases were less than two years' duration.

We are repeatedly asked if injection of the ganglion fails, will the major operation be rendered more difficult? Alcoholic injection does not interfere at all with subsequent operation.

We never advise an alcoholic injection into the ganglion without first explaining the advantages and disadvantages of the various types of treatment used for the relief of tic douloureux. We do not urge it in preference to the major operation in every case. The decision must rest with the patient. In old people with high blood-pressure, kidney disease, etc., we feel that alcoholic injection should be given preference over any other procedure.

In bilateral trigeminal neuralgia with a negative Wassermann, alcoholic injection offers more with less danger than the radical operation.

The technic which we employ is that devised by Hartel. Local anesthesia or in apprehensive nervous cases analgesia, obtained

by nitrous oxide or chloroform, is used. The patient is placed in the prone position with the head slightly elevated on a sand pillow. A stiff needle graded in centimeters, of slightly larger caliber than the one used by Patrick, is employed. The guard on the needle is fixed at 6 cm. from the point. The left index finger feels in the patient's buccal cavity through the narrow passage between the coronoid process and the tubercle maxillæ. The needle is introduced into the cheek about 2½ cm. from the angle of the mouth and 2 cm. below. With the guidance of the left index finger it is directed subcutaneously between the mandible and the maxillary tubercle directly upward until it meets with the hard, smooth resistance of the sphenoid bone. The finger may now be removed from the mouth. The needle at this time will be at a depth of 4 cm. Before any further manipulations are made, it is essential to keep in mind the fact that a line drawn vertically upward from the point of entrance of the needle into the skin will bisect the pupil of the eye if looked at from in front, while if looked at from the side it will bisect the articular eminence of the zygoma. The needle is now pushed backward and slightly upward, the operation becoming more painful as we approach the third branch. After several tentative feelings with the needle, the resistance suddenly ceases as we penetrate into the skull through the foramen ovale.

We no longer feel any bony resistance, but instead what is quite characteristic on long practice, the elastic-like resistance of the ganglionic tissue. The subjective symptoms of the patient vary greatly—some suffer excruciating pain while others do not complain at all. In cases which have previously received extracranial injections of the third division, some difficulty is encountered because of the fibrous alterations around the foramen. The penetration of this scar tissue gives rise to pain and makes palpation along the bone more difficult. In these cases we depend exclusively upon the direction of the needle with its relation to the pupil and the articular tubercle. A *trial injection, of a 2 per cent novocaine solution,* will determine whether the ganglion has been reached. In about two minutes, if we are successful, anal-

gesia occurs in the whole distribution of the trigeminus. If this action does not promptly follow, we are not in the ganglion, and experience alone will tell whether the injection should be repeated at a different depth or whether the needle should be withdrawn and the puncture repeated.

In 3 of our cases dilatation of the pupil occurred with the injection of novocaine. If our novocaine injection is successful and the anesthesia is confined exclusively to the area supplied by the fifth nerve, we inject the alcohol. The alcohol should be injected drop by drop, close attention being paid to the eye movements of the patient and his subjective symptoms. The cannula is cautiously moved from side to side in order to inject as much of the ganglion as possible. A total of 2.5 cc of 85 per cent pure alcohol is used. The cannula is rapidly withdrawn and the puncture wound painted with tincture of iodine.

For the first few days following the injection a careful record should be made of the extent of anesthesia obtained. If this area shrinks, *i. e.*, if we find sensation returning, particularly in the second division, we can suspect that the ganglion has not been sufficiently infiltrated to give a permanent cure. These are the types of cases where the pain is likely to recur. At times, this partial injection of the ganglion is indicated, but the patient must be warned that recurrence is more apt to occur.

Complications. The most common complication is a hematoma of the cheek. This yields rapidly to cold compress treatment. Paralytic keratitis has occurred in about 15 per cent of the cases reported. We have had one ulcer of the eye which healed up under treatment, leaving a slight scar and 1 case of severe keratitis. Septic meningitis has been reported by Hartel in 1 case. We have never seen this. The complications objected to by most men in criticizing the alcoholic injection are not due to the ganglion being infiltrated, but are due to the alcohol being injected elsewhere.

We regret to report the loss of one eye a year following the alcoholic injection. On account of the anesthesia of the cornea, the patient had been instructed to flush the eye daily with warm

boric acid solution. She mistook spirits of ammonia for her boric acid solution and flushed the eye with the former, with the deplorable results above noted.

If proper care is taken to observe the effects of the trial novocaine injections, most of the untoward results reported will not occur.

Our attention has been called to the paralysis of the motor root following the injection, and devices have been suggested by A. Bleichsteiner, of Vienna, to overcome the deviation of the jaw caused by this paralysis. We have only seen 1 case with motor palsy after six months. As a general rule, motor palsy clears up before the eighth month. Patients are unaware that it exists—it causes no inconvenience. Little or no atrophy of the muscles of mastication has been observed.

We are at the present time engaged in some experimental work on dogs, exposing the foramen ovale and injecting the ganglion with alcohol in order to study the late effects of alcohol infiltrated into the ganglion. We are not as yet ready to report on these, but are inclined to agree with the reports made by Otto May, of London, and Charles M. Byrnes, of Baltimore. Degeneration of the ganglionic cells with fragmentation and disintegration of the nerve fibers of the spinal root occur in all cases. We agree with them that one injection of alcohol will not destroy the ganglion, but it appears to cause a chemical division of the sensory root in a successful injection.

Cardiovascular Symptoms Associated with Goiter

BY PROFESSOR M. C. PINCOFFS
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(By Invitation)

An analysis of the cardiovascular symptoms associated with goiter shows that they may be divided into purely functional cardiac and vascular disturbances (tachycardia, peripheral

vasodilatation, etc.) which run more or less parallel to the basemetabolism, and another class of signs and symptoms (abnormal rhythms, increased diastolic pressure, cardiac enlargement) which point toward definite pathological changes in the heart and vessels. In goiter with hyperthyroidism cardiovascular symptoms vary according as to whether we are dealing with exophthalmic goiter or with adenoma of the thyroid with hyperthyroidism. In exophthalmic goiter the functional disturbances are most prominent and the evidences of organic damage occur, as a rule, only when the disease has persisted for some years. In toxic adenoma symptoms indicating organic cardiovascular lesions occur early and assume a dominating place in the clinical picture. Auricular fibrillation is particularly apt to occur in such cases.

In goiter without hyperthyroidism the heart occasionally suffers through mechanical impediment to respiration which disturbs the proper filling of the right auricle, and, through resultant pulmonary changes, increases the resistance in the pulmonary circuit. That simple colloid goiter exerts a harmful influence on the cardiovascular system is as yet unproven, but there is evidence tending to show that apparently latent adenoma of the thyroid may cause slow degenerative changes in the heart and vessels. The removal of all definite adenomatous nodules irrespective of manifest activity is advocated.

Some Comments Concerning Vitamins

BY WILLIAM EGBERT ROBERTSON, M.D.

Much is already definitely understood as to the etiological relation of vitamins to certain disease entities and syndromes, but we meet with many body disturbances which seemingly depend upon food factors about which we know as yet little or nothing, such as the asthenias associated with anorexia, perhaps

best illustrated by that peculiar condition called anorexia nervosa, or, going to the other extreme, and more easily dealt with, theoretically at least, the conditions resulting from excess food or excess of certain types of food, as seen in the obese, the potential diabetic, and perhaps I am justified in adding the hypertension patient. Yet when we come to apply what knowledge we possess, who can say that we can successfully prescribe for a case of hypo- or hypertension, or, granting the value of the water-soluble vitamins as appetite stimulants, we soon reach a state of disappointment and dissatisfaction, in adults certainly, less so in those whose growth is incomplete, when we attempt to reëstablish the balance of growth and nutrition.

To the practising doctor, no better instance of the value of food substances is presented than in the typhoid case. During the active infection, even though the patient be allowed and may take 2000 or 2400 calories a day, there is more or less progressive loss of flesh and strength, less, of course, than in those subjected to the former and really cruel plan of relative starvation. As soon as the active phase has subsided, however, convalescence and gain in weight are very rapid in those properly and hence sufficiently fed, and this is especially true of those to whom a liberal caloric diet has been given during the course of the infection. To bring home the importance of foods, both as tonics and reconstructives, I often say to patients, "You cannot buy good health in the drug store." This sometimes carries conviction and makes the problem of diet one more readily dealt with, for it cannot be denied that the public has not yet been educated to the importance of food in health or in disease. It is often astonishing how much can be done for the obese heart patient, with or without an endocarditis, by a sufficient but restricted diet. When it comes to the utilization of vitamins, however, except in a limited sense, we enter deep water.

To Casimir Funk we owe our knowledge of vitamins as specific food factors, though many had tilled the field before him and suspected their existence. He named them vitamins because he found them to be nitrogenous, amine-containing, and because he

found them to be of vital importance. He also gave us the terms avitaminoses or deficiency diseases, comprehensive names applied collectively to those diseases or syndromes arising from a deficiency of vitamins.

Beriberi was the first of the deficiency diseases to be studied physiologically and chemically, hence leading to the discovery of vitamins. The disease has been known to exist in Japan for over one thousand years, and in Brazil, quoting Lovelace, Funk says it had existed at least two hundred and fifty years. He says the disease occurred only occasionally in the warmer climates, and among rice-eating people, until toward the end of the nineteenth century, when the relative oriental isolation was forever disrupted by the penetration of the white race. Soon after the influx of Europeans, it was noted that beriberi spread in an amazing fashion, both in numbers and geographically. It was soon noted that among the natives, who clung to the hand-mill process of treating rice, the disease only arose sporadically, but with the introduction of machine milling of rice by the Europeans, where such rice was used as the staple diet, the disease spread rapidly and the mortality was considerable. It did not take long, when the above fact had been recognized, to determine that with the more thorough removal of the pericarp or outer layer of the rice, only ineffectually accomplished by the native hand-mills, and that for gustatory reasons, there was removed at the same time some substance of vital importance to the economy. The next step, of course, was to feed the removed bran to those affected, and the result naturally confirmed what had been previously only a suspicion. This was followed by experiments upon animals, and the missing link in the chain of evidence was finally and firmly applied. It is of further interest to note, and especially perhaps to those with socialistic leaning, that the owners of rice-polishing mills found that the bran possessed a food value of a very high order for cattle and commanded a high market price. This country, be it noted, was the first to take cognizance of these findings and to pass a law forbidding the sale of completely polished rice in the Philippines.

These facts established, it soon became known that there were other members of a group of substances now called vitamins, having in common a complex structure, nitrogenous, crystalline bodies, apparently belonging to a new chemical group and separable from the parent substances by appropriate precipitants. They are absolutely essential to life, are necessary only in small amounts, but may be given in large quantities without harm. It is also known that originally our store of vitamins is drawn from the vegetable kingdom. We know now that in the animal kingdom, certainly, an absence of vitamins in the diet means death and a deficiency means more or less departure from health. Besides true beriberi of the human we have the experimental type in birds, called *polyneuritis gallinarum*.

Scurvy and its close congener in children, Barlow's disease or Barlow-Cheadle disease, also experimental scurvy and pellagra (?), probably, too, rickets, osteomalacia, infantile spasmophilia, tropical endemic neuritis and sprue belong here. Manifestly various grades of symptoms may be present, some difficult of recognition, and then only when occurring in groups or when the character of food intake has been carefully investigated. On the other hand, such diseases as beriberi and scurvy, when at all marked, are simple to diagnose. When a limited diet has been taken uninterruptedly for weeks or months, a diet, for instance, consisting of white bread, flour, white rice, maize and cooked milk, the symptoms are many and variable, but for reasons now obvious their nature should be suspected. Among these, perhaps, multiple neuritis deserves the first place. The extent and distribution vary widely and the paralytic type may dominate, or, in other cases, spasmophilia and contractures. Other cases present a cardiac complex. Bradycardia is often an early symptom. The right side of the heart dilates, with dyspnea, cyanosis, diminution of urine volume, anasarca, hydrothorax, ascites and hydropericardium. These phenomena may occur in isolated, sporadic cases, but they are common in the well-developed case of beriberi. Then, too, the known, multiple hemorrhagic manifestations of scurvy, the swollen and bleeding gums, extravasations into and under the

skin, into the muscles, beneath the periosteum and serous membranes and the joint phenomena. Pellagra is diagnosed with its lesions in stomach and bowel, skin erythema and involvement of the central nervous system. Histories of war and pestilence, from remote times, cite instances of hunger edema, and occasional outbreaks have occurred in institutions and gaols. Sydenham, the father of medicine in England, makes reference to it. Finally, xerophthalmia, failing vision, with ultimate destruction of the eyes, has been traced to a lack of fat-soluble vitamins.

Foods in relation to lower animal life have a humane, economic and scientific interest. Steenbock and his co-workers have shown that cattle fed on roughage may develop osteomalacia, and that in such instances the food has been found to contain an excess of magnesium salts, which act as decalcifiers. This I quote from memory, not being able to recall the original reference. It is of importance, however, for confirmation should make us wary in ordering milk of magnesia, an especially common practice among pediatricians. It has been previously stated that, as a general rule, children are more susceptible than those of full growth to the milder types of avitaminosis and to errors in feeding in general. Another topic of most suggestive interest and value, I quote at some length from the article by G. Ennis Smith, which was published in *Endocrinology*, July to September, 1919. This topic is introduced in order to accentuate the importance of a study of foods in general, for it is not known to have association with vitamins in any direct sense:

"The author has described a thyroid disturbance that is prevalent among newborn domestic animals in western sections of this continent. The disease is one which affects swine, sheep, cattle, horses, goats and dogs, and occurs among such a wide range of animals that it appears very probable that if human beings were subjected to the same relative conditions a more or less severe form of a similar thyroid disturbance would result. A profound influence of pregnancy upon the maternal thyroid, which may develop into a pathological condition, has to some extent been recognized, but the attention has been confined mainly to the

maternal organism. The manifestations of the thyroid disturbance among domestic animals show very clearly, however, that the effects are more disastrous to the fetus than to the maternal organism. It is our opinion that when this subject has been thoroughly explored, many of the cases of infantile fatality and of arrested development of children, now vaguely designated as congenital, will be found to arise from a lack of function of the fetal thyroid which may be overcome by a proper diet and conduct of the mother during the gestation period."

It is of special interest to note that this malady is largely endemic and corresponds more or less closely in geographical distribution to those areas in which goiter is commonly found. Many of the young are stillborn and few live more than two days. The importance of this subject is best illustrated by the fact that the loss in Montana alone has amounted to about 1,000,000 young pigs annually, and, as previously stated, it is by no means confined to hogs. Symptomatically the condition resembles myxedema or cachexia strumipriva. The iodine content of the thyroid is low. The gland is hypertrophied—interesting in connection with the view of Hector MacKenzie that the gland undergoes hypertrophy in non-toxic goiter in an attempt to furnish more secretion. The evidence thus far favors the opinion that the disease is due to some deficiency of nutrition, "possibly an uneconomic use of available iodine." Hart and Steenbock have been able to produce hairless pigs presenting a symptom-complex quite like those the victims of this athyrosis by feeding pregnant sows a high protein diet, and were able to obviate it by iodine feeding. Though the pregnant sow shows little evidence of any ill-effect, there is some degree of maternal athyrosis; but the most malign influences are manifested in each succeeding litter.

Even more important, and more definitely related to these unknown substances which at present we call vitamins, was the experimental work of McCollum with young heifer calves. Three groups were fed in rations as similar as chemical analysis was able to determine but differing in their source. Group I was fed on corn; Group II on oats; Group III on wheat. Each group

was given the same amount of protein, carbohydrate, fat and salt. It required about a year before distinct differences were observed in the different groups. At that time the corn-fed animals were normal and their procreative functions were unaffected. The oat-fed animals were less sleek than the corn-fed, and their young, born fully two weeks before time, were stillborn or died soon after birth. Only one was saved by special measures. The wheat-fed were spare, rough coated, undersized, gave birth three to five weeks before term, and all the young died a few hours after birth, all being much underweight. Kept on the same diet, similar results ensued at the end of another year. Milk production for the first period amounted to an average of 24 pounds per day for the corn-fed, 19 pounds per day for the oat-fed and 8 pounds per day for the wheat-fed. These amounts during the second period of lactation were increased, averaging 28, 30 and 16 pounds respectively. This is not unusual in the experimental animals and even in the human, the mother suffering in direct proportion to volume and quality of milk produced.

Other experimental evidence, far too vast to be detailed in a brief paper of this character, has served to show the presence in certain foods of some substances vitally necessary to reproduction, to growth and to nutrition. Pure food mixtures containing all the primary food substances and necessary salts not only failed to maintain growth and nutrition, but in different animals produced different symptom-complexes. The earliest work was that of Eijkman, a Dutch chemist, who in 1897 first produced polyneuritis in birds by feeding them with polished rice. He found two or three weeks of such feeding sufficed to produce the disease, and that the addition of small amounts of the rice polishings sufficed to cure it. Curiously, he failed to grasp the full significance of his work, as did our American investigators, the Philippine Commission, whose work covered a period of over ten years, from 1900 to 1911. It was in 1906 that Hopkins, an Englishman, first called attention to hypothetical food substances which he called "accessory food factors." In 1907 Fraser and

Stanton, two Englishmen, were the first to find that curative virtue existed in the alcohol precipitate of rice polishings. In course of time it came to be shown that three and possibly four groups exist. McCollum has termed them fat-soluble A, water-soluble B and C, and more recently Hess has suggested the possibility of a D vitamin which may be antirachitic.

Mellanby, an Englishman, contends that the A or fat-soluble vitamin has antirachitic properties. This view, however, is not generally accepted by the American group of nutrition experts. Mellanby studied a number of fatty substances, notably cod-liver oil, butter fat, bacon fat, lard, suet and many vegetable oils. He also showed that cod-liver oil far exceeds all other fats in its antirachitic properties, but showed, too, that suet and butter fat both exercise an important influence on calcification. The vegetable fats and oils vary in antirachitic properties, many wholly lacking them, and even the more favorable ones possessing rather a small value. Mellanby also confirms the finding that by heat and oxidation of a fat much of its antirachitic property is lost. To Hopkins belongs the credit of first demonstrating that oxidation destroys fat-soluble A vitamin. Fat so treated loses the growth factor and the property of curing xerophthalmia, due to the absence of the fat-soluble A from the diet.

In their latest paper on this subject McCollum, Simmonds, Becker and Shipley, in the *Journal of Biological Chemistry*, 1922, liii, 293, in a paper entitled, "Studies on Experimental Rickets," have shown, first, that cod-liver oil when oxidized for twelve to twenty hours does not cure experimentally induced xerophthalmia. It is still able, however, to cause the deposition of calcium in the bones of young rachitic rats. Hence oxidation destroys fat-soluble A without destroying some factor which has the property of stimulating calcium deposition—bone growth and repair. Butter fat also possesses the property of depositing calcium, but to a much smaller degree than the fish oils. Mellanby showed also that cocoanut oil possesses antirachitic properties but not the antixerophthalmic. Hence it has been demonstrated that the best of all fatty substances is cod-liver oil and some other

fish oils, and that these possess preventive and curative properties against xerophthalmia, protecting against a deficiency of calcium and bringing about a deposition of calcium salts in rickets. It has also been shown that the antirachitic property is not due to the presence of fat-soluble A. Thus the proof of a fourth vitamin which seems to be able to regulate bone metabolism. In the same journal, of August, 1922, McCollum, Simmonds and Becker report another and less serious form of ophthalmia occurring in rats, due to a dietetic error, not lacking, however, in fat-soluble A. They do not feel that they have definitely established its etiology, but tentatively ascribe it to a diet high in chlorine, possibly high in sodium, while perhaps other inorganic salts may contribute.

Whatever may be the ultimate view as to the water-soluble substances, butter fat, which carries the A vitamin, is both nitrogen-free and phosphorus-free. It seems scarcely justifiable, therefore, to call this growth and nutrition factor a vitamin, yet custom has come to sanction it.

Animals do not synthesize vitamins, but obtain them directly from food eaten. It is obvious, therefore—as far as milk is concerned and also the flesh of cattle—that it is of vital importance to the welfare of the growing child especially that careful supervision should be given to the food substances of these animals. Fortunately for the adult human, Nature has been lavish in the distribution of the vitamins thus far recognized as such. The table of the British Research Committee gives the present knowledge concerning the vitamin content of foods. This was published some weeks ago in the *Journal of the American Medical Association*. It is too lengthy to give in full, but briefly, the A, or fat-soluble vitamin, is found in butter, milk, egg-yolk, carrots, spinach, lettuce, cabbage and the germ of cereals. A moderate amount is present in meat. The modern milling processes have deprived our grain foods of much of their essential food value, and it is appropriate here to call attention to the value of the unrobbed cereals as a food. This type is not affected by the usual methods employed in cooking. So, too, with the

B, or water-soluble vitamin, unless boiling is continued over a long period or a temperature above boiling is employed. Greater harm follows if the boiling medium is distinctly alkaline, a fact worthy of note in connection with the practice of adding an alkali, such as bicarbonate of sodium, to the water. Milk and, in greater amount, eggs furnish the B type, also condensed milk, cheese, most of the nuts, some of which contain A vitamin also, as the peanut, almond, cocoanut and hickory. The flesh of animals contains very little of the water-soluble B, but the brain, liver, kidneys and particularly the pancreas all contain it; carrots, cabbage, cauliflower and celery in abundance, likewise onions, parsnips and potatoes. Spinach is rich in both A and B, and the added value of the vegetables is that most of them contain a large amount of antiscorbutic properties. This is also true of most of the fruits. In beans and peas the vitamin is found throughout their substance, but in grains it is practically confined to the embryo. Yeast is rich in water-soluble B vitamin; it lacks both the A and C type. It is probable that it serves to stimulate the appetite, but Rabelais probably hit the nail on the head when he said, "The appetite comes with eating." So, too, said Shakespeare, "Good digestion waits on appetite, and health on both." It is even more likely that the use of B vitamin foods would serve infinitely better than yeast to awake a desire for food.

It is surprising how many people confine their diet to relatively few articles of food and, despite the so-called "tonics," continue to take insufficient nourishment—in my opinion, chiefly because they lack the necessary variety and the stimulus to appetite which I believe is given by water-soluble vitamins. That appetite really does come with eating is not sufficiently recognized. I cannot feel that medicinal tonics are necessary. The water-soluble C vitamin, antiscorbutic, is very readily destroyed by heat, drying and alkalies. This is found especially in oranges, lemons and grapefruit, less in limes, with which apples and pears are classed, but in large amount in cabbage, turnips, tomatoes, lettuce and watercress.

At present it is a matter of some difficulty to adjust diets with

any sense of conviction in cases of nutritional disturbances, whether in the stage of growth or in later life. So many factors have to be dealt with, over some of which, as individuals at least, we have no control. Indeed, it is a question whether the Pure Food Law has been an unmixed blessing. Possibly our legal representatives at Washington may decide these questions for us, as they have done in the case of alcohol, narcotics and in the revision of the *Pharmacopœia*.

In the choice of food for the average adult, enough is now known to make certain the statement that the mere presence of proteins, carbohydrate and fat will not necessarily suffice even when in correct proportions. Certain it is that accessory food factors are necessary. This is of special importance in the case of the nursing mother, and just as important in cattle, since milk is the staple food of the infant. Vitamins, not being synthesized, can only be gathered from food intake. The diet, especially that of nursing mothers, should therefore contain a variety of the green vegetables especially, despite the common prejudice. The French empirically have long known what they are doing when they put the water in which fresh vegetables have been boiled into their soups. In addition, of course, both A and C vitamins are necessary, but appetite will usually increase under the stimulus of B vitamins. Hand-reared babies are safe on pasteurized milk as far as A and B vitamins are concerned, but the sensitiveness of C vitamins to heat and alkalies should suggest the addition of orange juice or even tomato juice to their diet, for that is well borne. Spinach, which contains a large amount of A, B and C vitamins, is not only an excellent food for adults, whether nursing mother or not, but the expressed juice is decidedly advantageous for nursing children, and especially for bottle-fed babies. Both adults and children should be systematically weighed and a record kept. Cod-liver oil, rich in A vitamin, is now regarded as the best remedy in rachitic children. Whether, as Mellanby believes, its virtue is due thereto, or whether it possesses another factor, tentatively referred to as D vitamin, is still in doubt. (The contribution of McCollum and his co-workers, quoted above,

added since this paper was written, has apparently settled this point.) It is said, too, that it possesses the property of fixing calcium salts. Schmorl has called attention to the seasonal development of rickets, being more frequent in winter than in summer. This is of interest in connection with the recent claim of Hess that the disease is favorably influenced by sunlight and also by the ultraviolet ray.

It is still a question as to whether pellagra can be classed with the avitaminoses, though there is considerable suggestive evidence that it depends upon some food factor as yet unknown. Time does not permit the inclusion of many interesting experimental contributions to this subject. Suffice it to say that rats are not subject to scurvy, while guinea-pigs are. In a recent number of the *Journal of Biological Chemistry*, Shipley, McCollum and Simmonds report that rats fed on a complete diet, but for the absence of the antineuritic water-soluble B, develop bone changes exactly like those in guinea-pigs fed on a complete diet lacking only the antiscorbutic factor. On a diet lacking in A vitamin their bones become osteoporotic. Clinically, too, in the human, beriberi and scurvy have several lesions in common. The lesson to be deduced is that one should not place reliance on a single food factor, basic or accessory, but should endeavor to utilize a complete food in every sense in dealing with depraved nutritional states.

In closing, I quote from an article in the *Scientific Monthly* for October, 1919, by Prof. I. Newton Kugelmass, head of the department of chemistry, Howard College:

"Educational reforms will bring the science of nutrition from the theoretical background to the practical front. For that indispensable service we need universal instruction in nutrition, dietetics, prophylaxis, child health; competent, trained elementary school teachers, and health visitors in nutrition and public health; infant-welfare centers; social-service staffs; more social-unit organization work under the auspices of the child-welfare leagues and child-health associations; public coöperative kitchens; public demonstration centers; public-school child-feeding; regular

and periodic inspection of school children; more financial aid to mothers; more housekeepers' clubs; more public lectures on health, nutrition and sanitation. The public cannot go to the schools; we must go to the public."

FEBRUARY 26, 1923

Hemotoxins from Intestinal Parasites

BY JOSEPH LEIDY, M.D.

That certain parasitic worms secrete substances that affect the blood of their hosts deleteriously has been shown conclusively by the researches of Swartz, Schaumann, Tallqvist and others.

The broad tapeworm of man, which is known to cause severe anemia, contains a hemolytic agent, according to the experiments of Schaumann and Tallqvist. Hookworms secrete a hemolysin and an anticoagulin, according to Calmette and Breton (1905) and Loeb and Smith (1904). The whipworm, Trichuris, secretes a hemolysin: Whipple (1909) and Garin (1913). In 1865, Kuttner cites a case of blood destruction cured by expelling ascarides: Filatoff (1897). Karaven cured a case of pernicious anemia by expelling ascarides from the intestines. François (1906), in the course of his investigation on anemia of miners, found many cases of severe anemia in which hookworms were not present but which showed numerous ascaris eggs in the feces, and according to Schwartz, hogs and horses infested with A. lumbricoides bear out the fact that symptoms of anemia are frequently associated with such infections.

Two views as to the cause of parasitic anemia have been held by different observers: One, that the anemia results from depriving the host of blood by sucking and the other through the secretion of a toxin, hemotoxin, hemolytic in character.

"Owing to the fact that the direct abstraction of blood by parasites appears to be inadequate as an explanation of the causes

of anemia in parasitic disease, and in view of the fact, in tapeworm infections, which are accompanied by anemia, due entirely to the presence of the parasites, the direct abstraction theory is inapplicable, from the researches, particularly of Schwartz and others, the view that hemolysins from parasites are of etiological significance in parasitic diseases appears to be entirely justified" (Swartz).

A brief review of some observations and researches leading up to these conclusions is not without interest and importance. The observation of Kuttner (1865) referred to by Huber (1870) expressed the opinion that many symptoms put down to roundworms are caused by a peculiar irritating matter which they contain.[1] Huber draws attention to an observation of Mirams that the examination of A. megalocephala, had twice caused him most unpleasant symptoms, such as sneezing, swelling of the puncta lacrimalis, hypersecretion of tears, with violent itching and swelling of the fingers. Huber himself states, after examining twelve examples of A. lumbricoides, suffered from troublesome itching of the hands and neck; over the latter the skin was raised here and there in lumps, smaller lumps appearing on the forehead. His right ear swelled up and for an hour a plentiful secretion came from the meatus associated with a most unpleasant sensation of pulsation in the right side of the head, radiating from the ear, the conjunctivæ became inflamed, accompanied with severe itching; the inflammation leading to chemosis in the right eye and finally itching of the hands. After the lapse of an hour these symptoms gradually subsided. Huber considers that these symptoms were caused by a peculiar substance which gives off the strong smell peculiar to roundworms, and which, as Leuckart supposes, is contained in the vacuoles between the muscular fibers. In 1898, Schaumann and Tallqvist reported the discovery of a blood toxin in the broad tapeworm of man, but little attention was paid to this observation. Schimmelpfennig (1902) announced that the symptoms produced by the horse ascaris were due to a hemotoxin, he observed in the presence of

[1] Ziemesen, Encyclopedia, vii, 741.

the coelomic fluid of ascaris equorum, red blood cells from the horse became crenated and were ultimately destroyed through a process hemolytic in character. The specific gravity of the blood nor the percentage of alkalinity is given in this case.

Anemia has been frequently associated with the ascarides in both man and animals and the literature reveals the fact that this anemia has been mistaken for hookworm and pernicious anemia.

The researches of Faust, Beumer, Dascotte (cited by Weinburg), Calimida and more recently Ransom, and Kolmer, indicate the importance of a closer study by clinicians of these problems, which have been confined more particularly to medical and economic zoölogists.

It has been shown that Dibothrium latum is capable of causing severe anemia in man, clinically indistinguishable from pernicious anemia, and according to these investigators differing from the former in one respect only, namely, by the disappearance of the symptoms and recovery of the patient after expulsion of the parasite. On the other hand there are numerous cases on record in which the presence of the D. latum in man was not accompanied by anemia. What then is the explanation of the immunity noticeable in one group of cases not present in the others? Certain it is, parasites secrete toxic products. Swartz has shown that the serological reaction of the hosts harboring parasites afford proof that parasitic worms liberate products against which the hosts develop defense or immunity reactions.

Worms belonging to the genus ascaris contain a hemolysin which is closely bound to the muscular tissues, as indicated by Leuckart, and is therefore but slightly soluble in water. Others are of the opinion the toxin is given off from the intestinal canal and other structures and organs of the parasites, which fact may account for the negative results obtained by other investigators.

The ascarides also appear to secrete a feeble anticoagulin. In a series of experiments *in vitro*, Swartz found that the extracts of the intestines of the parasites were strongly hemolytic, whereas

extracts of the body wall showed no hemolytic effects. Extracts of the reproductive organs were but moderately hemolytic. In a second series of experiments extracts of the intestines were strongly hemolytic, whereas extracts of the reproduction organs and body wall showed weak hemolytic powers. Leidy[1] has shown that parasitic worms harbor their own parasites both upon the exterior and interior of the animal, and Weinberg has in fact described what appears to be a disease in worms belonging to the genus ascaris, which is characterized by the presence of certain pigmented spots that are clearly visible through the cuticle. Swartz has also observed the condition in specimens of A. lumbricoides.

The present note has to do with the results of a clinical study of 3 cases of parasitic infestation and point strongly to the hemotoxin of ascarides as an etiological factor deserving of attention.

Case I.—A case of corneal ulcer, under the care of Dr. E. S. Saylor. This patient, a subject of corneal ulcer, several years previously, was seen by the reporter for the first time in April, 1921. During the previous attack the illness covered a long period and proved resistant to treatment, when parasites were accidentally noticed in the stools. The use of a vermifuge, followed by the expulsion of a large number of roundworms, resulted in rapid and complete recovery. The patient was referred to the writer as a possible case of incipient tuberculosis. The physical examination proved negative. The mother volunteered the information, during the previous attack, that the child was "troubled" with worms. Acting upon this suggestion, examination proved the patient to be the subject of intestinal parasites. Upon the administration of a vermifuge the patient expelled a coil of twelve roundworms, followed in twenty-four hours by the expulsion of a mass of others, dead, and having the appearance of being partially digested, and in a state of decomposition.

[1] Leidy: Flora and Fauna within Living Animals, Proc. Acad. Nat. Sci., Philadelphia, 1850.

The blood examination showed hemoglobin 70 per cent and eosinophiles 14 per cent.

From the day following the expulsion of the parasites the patient began to show marked physical improvement and the corneal condition subsided rapidly. Examination of the feces showed the absence of ova from the stools. There has been no recurrence.

Case II.—Patient, aged nine years, with chronic eczema of eight months' standing; occasional epistaxis and marked malnutrition. The patient had run the gauntlet of local and tonic remedies and the parents were in despair. The general physical examination proved negative. Microscopic examination of the feces showed the presence of large numbers of the ova of ascarides. Eosinophile count 16 per cent and hemoglobin 70 per cent. Following treatment the patient expelled twenty roundworms; in one coil there were three dead and disintegrated or partially digested worms; the general condition of the patient showed marked improvement from the expulsion of the parasites and absence of ova from the feces, complete recovery continued without interruption.

Dr. T. E. Brown, in *Johns Hopkins Hospital Reports*, refers to a case of chronic eczema with an eosinophile count of 22 per cent, but no reference is made to the condition of the gastrointestinal or the results of treatment.

Buckle, working under Leichtenstern, has established the interesting fact that all varieties of helminths from the harmless oxyurides to ankylostoma, may bring about an increase in the eosinophiles, showing in oxyurides as high as 16 per cent; ascarides, 19 per cent; ankylostoma, 72 per cent; tenia mediocanellata, 34 per cent. (Ehrlich and Lezarus.)

The rapid recovery from both these attacks, following the expulsion of the parasites with dead and partially digested and decomposed worms in the second attack, and the return of the eosinophile count, 2 per cent to normal, must be viewed as more than a coincidence. The child has had no further illness or indisposition, and is in vigorous health.

Case III.—During March, 1912, I saw in consultation with Dr. R. E. Brown, of New York City, a male child, aged six years, the subject of frequent attacks of angioneurotic edema. The patient had been the subject to attacks at intervals during the previous six months.

The clinical picture was characteristic, with swelling of the face, eyelids and scrotum, large wheals over the back and legs, with croupous respiration, the symptoms at times alarming.

Blood examination: Hemoglobin 80 per cent and eosinophiles 10 per cent.

The group of symptoms presented is not uncommon during disturbed intestinal metabolism, intestinal toxemia, and attention was directed to the gastrointestinal tract as the possible focus of the condition.

The urine showed no increase in indican nor was there increase in the excretion of sulphoethers.

Microscopic examination of the feces showed large numbers of the ova of ascarides.

Santonin was administered and followed promptly by the expulsion of twenty-six worms, four dead, disintegrated and partially disgested. Recovery was prompt.

There has been no recurrence in ten years.

Intestinal toxemia is not infrequently an etiological factor in cases of angioneurotic edema, but in all those cases which have come under the observation of the writer the indican content of the urine ran high, with an appreciable increase in the elimination of the sulphoethers, and in no case was there an increase in the eosinophile count. The recovery under treatment of a diet rich in carbohydrates is usually prompt and effective.

These 3 cases appear of sufficient interest to place on record as indicative of the existence of a hemotoxin from parasitic infection to account for the symptoms, particular attention should be given to the existence, in each case, of dead, partially digested and decomposed parasites among the living as playing a part in accounting for the profound toxemia.

Taken in connection with the experiments on the action of the extracts of parasites *in vitro*, the clinical and laboratory picture present features of special interest.

Conclusion. All cases, especially among children, presenting malnutrition and anemia with increase in eosinophiles, should be looked on with suspicion as being the subjects of intestinal parasites and microscopic examination of the feces for ova, and parasites should be the routine of all clinical laboratories. Indeed, the time has arrived when no clinical examination can be considered complete without a microscopic analysis of the feces.

In any health survey of our school children, examination of the feces should become a routine part of the physical examination, as was pursued in our army camps during the World War.

The Treatment of Diabetes

BY ALFRED STENGEL, M.D., AND LEON JONAS, M.D.

In reviewing the development of the modern treatment of diabetes, one can readily distinguish three distinct periods:

The first of these dates from the time of Claude Bernard's researches on glycogen and the functions of the liver. Though many advances were made during this period and much was learned regarding the chemistry of the disease and the bearing of this on the all important condition—diabetic coma—it is quite clear that nothing of a fundamental sort was learned regarding the treatment of the disease. The withdrawal of sugar from the diet naturally first suggested itself; later, with more definite knowledge of carbohydrate metabolism, restriction of starches was also practised, but the practically unlimited meat-fat dietary which resulted was notoriously inefficacious. Excepting in certain mild diabetics the treatment of the disease accomplished little or nothing, and probably, in many cases, hastened unfavorable conditions. Nevertheless, certain facts were gradually accumulating as a result of

chemical studies of the blood and urine or, in some cases, as a result of accident. In the former of these categories may be placed the practice of feeding large quantities of carbohydrate food and alcohol in cases in which coma seemed imminent. In the latter may be put the discovery of the value of green days or fasting days, such as Van Noorden advised in connection with his oatmeal cases. This practice was entirely empirical. Possibly, also, the occasional advantages derived from the rice cures, potato cures or milk cures may have been due to the reduction of the total caloric intake incident to these methods. No one in this early period appreciated or emphasized the possible value of systematic underfeeding.

The method of Allen, which inaugurated and comprised the second period, began about 1915 and continued down to the present date. That this was a real advance became immediately evident to all of us who had considerable clinical experience in the earlier period, and who had, therefore, a full realization of the essential inefficacy of the methods of treatment in vogue. With the introduction of Allen's method we were enabled to render cases of mild or severe diabetes sugar-free in a manner that was dramatically impressive and contrasted most favorably with the unsuccessful practice of the past. Even in cases with actual or threatened acidosis the results were often most gratifying. The immediate effect of this almost startling result of a new method of management of the disease was to create an altogether unwarranted hopefulness of permanently controlling its advance and perhaps of curing it outright. No one familiar with the underlying pathology of diabetes could, on careful reflection, have felt confident that the results of this method would fundamentally influence the progress or continuance of the disease; but it is not unfair to say that experienced clinicians were so encouraged by the prompt control of glycosuria, and even of acidosis, that they experienced extravagant hopes of the ultimate effects of the method. Time, however, disillusioned even the most sanguine. While undoubtedly patients were temporarily rescued from the unfavorable effects of glycosuria and hyperglycemia, and

even of acidosis, further continuation of treatment along the lines of reduction of diet soon demonstrated that to remain sugar-free, the diabetic—at least the severe one—must reduce his diet more and more; and while those who pursued the method assiduously had the satisfaction of reporting their cases sugar-free over considerable periods of time, this result was achieved by successive curtailments of diet and greater and greater deficits in nutritional requirements. It is undoubtedly true that in certain cases of diabetes the reduction of blood sugar, the elimination of glycosuria and general improvement in metabolism established conditions favorable to a restoration of function and, unquestionably, some cases recovered a certain increased capacity for carbohydrate by the rest secured through this method, but such increased carbohydrate tolerance was less satisfactory than was hoped for, and was often wanting in severe cases. In the majority of cases greater and greater restrictions of diet were attended by increased loss of weight and general loss of vitality without evidence of any essential control or cure of the disease.

Meanwhile, during the first and second periods, increase of knowledge of the fundamentals of carbohydrate metabolism was accumulating, and the investigations of Minkowski and, later, those of Opie had shown that the pancreas and especially the islands of Langerhans are the localities where the controlling secretions are elaborated. It was clear to all who studied the disease that the pancreas is the source of an internal secretion that governs carbohydrate metabolism, and various attempts were made to relieve the disease by administration of raw pancreas, pancreatic extracts or the pancreatic ferments by mouth or hypodermically, but without effect. Unsuccessful attempts at obtaining the internal secretion were made by a number of investigators until Banting's experiements proved successful.

The third—the present—period in the treatment of diabetes begins with the investigations of Banting, and the preparation now available for treatment—insulin—has been shown positively to represent the internal carbohydrate-controlling secretion of the pancreas. Experimental and clinical experiences in sufficient

number have accumulated since the preparation has been available for study to show that insulin injected into animals or man acts in the same way as the normal internal secretion of the pancreas. The effect in cases of diabetes has been to facilitate carbohydrate metabolism and to restore artificially the utilization of carbohydrates. It is not too much to expect that, with the use of this remedy, normal metabolic conditions may be maintained in the diabetic, and that under these circumstances the strength and nutritional condition of the individual may be held intact until the pancreas, by rest, may recover its lost function. How frequently, how completely, and in what type of cases this artificial rest of pancreatic function will be followed by restoration of anatomical and functional integrity is a matter future experience must decide.

The cases selected from our series at the Hospital of the University of Pennsylvania illustrate the therapeutic effects of insulin on severe cases of diabetes.

Case I.—Insulin 5. M. I., white, female, American, aged thirty-four years, admitted November 16, 1922. Two years ago she began to have polydypsia and polyuria and gradually lost weight. On admission she weighed 91 pounds. She was untreated until November 12, 1922, when a physician was called to treat an acute gastrointestinal attack. A diagnosis of diabetes was made and the patient immediately put on a restricted diet. On November 14 respiratory distress developed. On the evening of November 15 she became delirious and then stuporous. She was admitted to the hospital in the afternoon of November 16.

On admission she was in coma, from which she could not be aroused. There was marked respiratory distress. She was given intravenously 500 cc of a solution containing 5 per cent of glucose and 2.5 per cent of bicarbonate of soda. One hour later blood was taken from a vein at the elbow for analysis. The CO_2 capacity was 17 volumes per cent; the blood-sugar concentration. 0.411 per cent. The urine showed a marked glucosuria and ketonuria. After the venous puncture 20 units of insulin were

PROTOCOL NO. 1

Day ending.	Diet per day.				24-hour urine.				Plasma.		Blood.		Insulin units per day.
	Protein, gm.	Fat, gm.	Carbo-hydrates, gm.	Calories.	Glucose, gm.	Acetone.	Diacetic acid.	Total N, gm.	CO_2 volume, per cent.	PH.	Ketones, gm. per liter.	Glucose, per cent.	
11–16	..	..	..	....	xx	xxx	xxx	....	*17	....	....	.411	3
17	..	..	..	....	6.8	xxx	xx	....	...	....	1.27	.265	32
18	..	..	..	....	....	....	...	....	†41	7.34	....	.313	32
19	..	..	..	....	20.0	xx	x	....	...	....	....	....	32
20	..	..	..	....	2.9	xxx	x	....	†66	7.50	0.18	.200	24
21	41	31	32	476	26.4	8.42 gm.	...	5.92	...	....	....	....	16
22	45	31	29	559	40.2	xxx	xx	....	....	....	....	....	16
23	45	27	25	526	3.9	xxx	xxx	....	†29	7.24	0.84	.174	32
24	43	14	34	460	tr.	x	0	....	†60	....	....	....	32
25	40	33	29	569	....	....	...	....	†73	7.48	....	.035	32
26	41	27	60	672	5.7	0	0	....	...	....	....	....	32
27	46	28	81	797	4.9	x	0	4.20	...	....	....	....	32
28	48	29	88	832	tr.	x	0	....	...	....	....	.094	32
29	53	73	85	1239	....	....	...	....	†71	7.54	....	....	32

* = capacity. † = content. xxx = strong reaction. xx = moderate reaction. x = weak reaction.

PROTOCOL NO. 2

Day ending.	Diet per day.				24-hour urine.				Plasma.		Blood.		Insulin units per day.
	Protein, gm.	Fat, gm.	Carbo-hydrates, gm.	Calories.	Glucose, gm.	Acetone.	Diacetic acid.	Total N, gm.	CO_2† volume, per cent.	PH.	Ketones, gm. per liter.	Glucose, per cent.	
2–12	28	8	269	1266	120	xxx	xxx	18.5	*16	*6.98	*0.370	*.326	30 } 10 units
13	27	14	122	733	29	xx	0	....	44	7.32	0.110	.306	40 } every 6
14	35	18	147	904	84	xx	0	10.4	64	7.51	0. 74	.153	40 } hours.
15	42	16	99	720	36	xx	0	....	58	7.42	....	....	30 } 10 units
16	39	20	39	498	8	x	0	8.12	58	7.43	....	.190	30 } before
17	47	20	79	702	tr.	x	0	....	...	....	....	....	30 } each meal.

* = before insulin. xxx = strong reaction. xx = moderate reaction. x = trace. † content.

given hypodermically. Within two hours she could be aroused. Ten units were given five hours later. Protocol No. 1 gives the progress of the case during the first fourteen days of her stay in the hospital.

The analysis of the protocol shows that relatively small doses of insulin may rescue a patient from coma. The shift of the acid base equilibrium was most striking. In four days it passed to the alkaline side of normal. The urine was alkaline at the height of the shift. On the eighth day the plasma reaction again changed to the acid side of normal following a reduction in the dosage of insulin because of an inadequate supply. After the dosage of insulin was increased the reaction passed in forty-eight hours to the alkaline side. The quantity of ketones in the blood fluctuated with the shift of the acid-base equilibrium.

On the morning of the tenth day the patient again became somewhat stuporous. There was no evidence, however, of Kussmaul breathing and analysis of the blood and urine showed a picture quite different from that of coma. Urine obtained by catheterization showed no sugar or ketone. The blood-sugar concentration was 0.035 per cent. About 25 grams of glucose were given at once and she recovered in a few minutes from the stupor. When discharged, December 22, 1922, she was receiving 8 units of insulin before each meal with 80 grams of carbohydrate per day, and remaining aglucosuric and free of ketone. At the present time, seven months after discharge, on 10 units of insulin twice a day—before breakfast and supper—she is able to carry on her household duties.

Case II.—Insulin 11. T. C., white, male, American, aged eighteen years, admitted February 11, 1923. The onset of diabetes was in February, 1921, after an attack of mumps. With the exception of a few weeks of hospital treatment in July, 1921, he had been untreated. On February 6, 1923, he developed acute tonsillitis, which rapidly brought about the present crisis. On admission it was difficult to arouse him. He presented a classic picture of severe diabetes. Air-hunger was marked. Examination

of urine showed sugar and much diacetic acid. Protocol No. 2 presents his progress during the first six days in the hospital.

The outstanding features of his case are the plasma pH 6.98, which up to this time is the lowest, we believe, recorded with recovery, and the rapid shift of the acid-base equilibrium from markedly increased acidity to the alkaline side of normal. At the outset 10 units of insulin were given every six hours. After two days the dosage was reduced to 30 units per day.

On March 19, because of a break in diet, acidosis developed. The pH of the plasma passed to 7.22 and the CO_2 content to 38 volumes per cent. Ten units of insulin every sixth hour for three days restored his acid-base equilibrium to normal. He was discharged from the hospital, May 21, 1923, sugar-free and ketone-free on 10 units of insulin three times a day with 80 grams of carbohydrate per day and a total caloric intake per day of 1700 calories.

Preparation of Pneumococcus Antibody Solution[1]

BY FRANK McELROY HUNTOON, M.D.

(By Invitation)

Since the beginning of the specific biological treatment of disease by means of the introduction into the patient of animal sera containing immune bodies or so-called "antibodies," the effort has been made to not only concentrate such immune bodies but to eliminate from the sera employed those portions which are not therapeutically active and which may produce disagreeable or dangerous reactions. Such efforts have usually been confined to the so-called "salting-out" procedure, which, owing to its nature, can never produce a pure solution of the antibody content free from the serum proteins.

In 1902, Landsteiner, working with a red blood cell agglu-

[1] From the Mulford Laboratories.

tinating serum, demonstrated that if the immune body has combined with the antigen and the extraneous serum removed by washing, that the digestion of the sensitized antigen would enable the recovery of a certain amount of the antibody in an active form, and such solutions showed, with the ordinary tests, an absence of protein. Later, various observers demonstrated the same principle, working with various antigens and antibodies, both *in vivo* and *in vitro*.

In 1915, Chickering utilized the known phenomenon that during the precipitin-precipitinogen reaction, most of the antibodies present were thrown down or adsorbed with the precipitate formed, and that such precipitates could be dissolved in alkaline solutions with the recovery of the antibody content. This observer purified antipneumococcic serum in this manner, but since the precipitate in such cases is in most part derived from the serum proteins, such recovered solutions necessarily contain serum protein and have the same clinical disadvantages as serum.

In 1919, our own work was started on this subject, utilizing the principle demonstrated by Landsteiner and others, and applying these principles to the attempt to produce a solution of pneumococcus antibodies which would be completely or practically free of serum protein, and with a concentration of antibody sufficient to justify its employment in the treatment of pneumococcus pneumonias.

It is unnecessary to go into the large amount of experimentation that was necessary before this practical result was produced, but we can indicate at this time the fundamental procedure which is now the basis of our technic and show somewhat more in detail the character of the solutions obtained.

The serum employed is a pneumococcus horse serum containing immune antibodies against the three fixed types of pneumococci. To this serum is added a large number of pneumococci of the three fixed types. These are allowed to take up or adsorb all of the antibody that they will. The bacteria are now thrown out by means of a centrifuge, carrying the attached antibody with them. The sensitized bacteria are washed with salt solution to remove the gross amounts of serum that cling to the organisms, and

are finally treated by immersion in a mildly alkaline salt solution and are then heated to 55° C. over a period of from thirty minutes to one hour. During this heating a certain amount of the adsorbed antibody is removed from the bacteria and goes into solution. Centrifugation is again employed and the supernatant fluid, which contains antibody in solution, is passed through an earthenware candle for purposes of sterilization. The solution so obtained is the one with which we are at present dealing. This is a water-white, perfectly clear solution, foaming slightly on shaking, containing 0.85 per cent salt, 25 per cent sodium bicarbonate, a small amount of bacterial protein and the antibody content.

The two questions to be answered as to the availability of such a solution are:

1. The potency: This solution, when properly prepared, contains sufficient antibody so that a 0.2 cc dose protects white mice against one million fatal doses of a virulent Type I organism. This is the minimum standard which has been adopted. Some lots will do much better than this.

2. The second question is the presence or absence of serum protein and other protein in this material. Tests for the presence of nitrogen have shown that these solutions contain a very small amount of nitrogenous material. We have failed to demonstrate the presence of serum proteins by any of the more delicate chemical tests. However, an examination by the biological method, the production of the anaphylactic state in guinea-pigs, has shown that 5 cc doses will, in a certain number of guinea-pigs, produce the sensitive state, but not in all. The theoretical dilution of horse serum in these solutions would be about 1 to 1,000,000, and this corresponds fairly closely with the results obtained with the guinea-pigs.

Also, we have the results in the treatment of over 600 cases of pneumonia where there has been in no instance symptoms of serum sickness or anaphylactic shock.

During the winters of 1920-21 and 1921-22 this material has been used extensively in the treatment of lobar pneumonia. Such results as have been obtained will be given here tonight by Dr. Cecil.

From the laboratory viewpoint, we can say that the clinical results have confirmed the laboratory findings, that we have here obtained a solution therapeutically active in this disease, and which is free from the objections (anaphylactic danger and serum sickness) which are inherent in the use of serum.

The material produces, on intravenous injection, a reaction simulating that of the so-called "foreign protein" reaction. The cause of this reaction is not at present understood, and as it is a clinical phenomenon, I prefer to allow it to be discussed by Dr. Cecil.

In closing, I wish to emphasize that any success which we may have had is due to the close and hearty coöperation of the various institutions which have been interested, and the scientific enthusiasm of the men concerned in the actual practical testing of the value of the material.

The Treatment of Lobar Pneumonia with Pneumococcus Antibody Solution

BY RUSSELL L. CECIL, M.D.
NEW YORK

Since the work of Cole and his co-workers at the Rockefeller Hospital, Type I antipneumococcus serum has come into fairly extensive use in the treatment of Pneumococcus Type I pneumonia. No specific treatment of any kind has been worked out for the other types of pneumococcus pneumonia, and even in the case of Pneumococcus Type I infections, serum treatment has not proven to be altogether practical for the general practitioner. The chief reason for this has been the difficulty and delay connected with the proper typing of sputum.

During the last two years we have been working with a modified antipneumococcus serum at Bellevue Hospital. This preparation was first worked out by F. M. Huntoon, of the Mulford Laboratories, and is nothing more than a water solution of the immune bodies originally contained in polyvalent antipneumococcus

serum. By a very ingenious procedure, Huntoon was able to extract the immune bodies from the antipneumococcus serum and redissolve them in water. This product is known as pneumococcus antibody solution, and contains protective substances against Pneumococcus Types I, II and III.

The advantages of this preparation are:

1. It is polyvalent—that is, it is aimed against the three fixed types of pneumococcus—and according to our figures, it has also shown itself to be of distinct value in the treatment of Type IV pneumonia. By reason of its polyvalent character, typing of the sputum is no longer absolutely necessary, and treatment of the patient can be instituted as soon as the diagnosis of lobar pneumonia has been made. This, of course, is a great advantage over serum.

2. Pneumococcus antibody solution is practically free from horse protein, hence there is no danger of anaphylaxis or serum sickness. This removes a great source of worry for the physician and discomfort to the patient, and there is therefore no reasonable limit to the amount of antibody solution that can be administered.

3. Pneumococcus antibody solution contains a certain amount of pneumococcus protein, which may conceivably act in the capacity of a vaccine.

4. The fact that the preparation is a water solution renders it very soluble and easy to absorb and possibly presents the immune substance to the body in a form more acceptable than that of serum.

The antibody has usually been administered intravenously in doses of 50 to 100 cc, the technic being similar to that of serum administration. Injections are given once or twice, sometimes three times a day. The reaction produced by antibody is very characteristic and striking. In twenty to forty minutes after the injection the patient usually begins to shiver and is soon in the midst of a hard chill which lasts from fifteen to twenty minutes. At its conclusion the patient complains of fever and the temperature may have risen to 106° F. or even to 108° or 109°. This high temperature usually persists for only a short time, thirty to

sixty minutes. The rapid fall is accompanied by a profuse perspiration which may continue for several hours. The fall in temperature may be slight but is often extensive, amounting to 8° or 10°. It may be temporary or permanent, depending on the stage of the disease. These reactions are very similar to the so-called "foreign protein" reaction, and are probably due to a disturbance in the colloid equilibrium of the blood. The dose of antibody administered does not bear any close relation to the character or severity of the reaction. In one patient 10 cc may produce a severe chill, while another patient may tolerate 100 cc without any reaction whatever. The reactions are more severe when treatment is started late in the disease.

During the past two and a half years this solution has been tested on a large scale in the wards of Bellevue Hospital. Through the kind coöperation of the various directors all cases of lobar pneumonia admitted to the twelve medical wards were studied bacteriologically, and in six out of the twelve wards practically every case of lobar pneumonia was treated with intravenous injections of pneumococcus antibody solution. The other six wards were used as control wards. In these wards, patients with pneumonia received no antibody, but in other respects were treated practically the same way as the patients who received antibody. Specific treatment was started as soon as the diagnosis of pneumonia was made. The results of this experiment are shown in Table I

The results of treatment with antibody were much more striking in cases that were treated early in the disease. For example, in Table II it will be noticed that the death rate for Type I pneumonias treated during the first forty-eight hours of the disease was only 8.9 per cent, while controls admitted to the hospital during the first forty-eight hours of the disease showed a death rate of 23 per cent. Similar reductions are noticed in the other types. Altogether, 114 pneumococcus pneumonias treated with antibody during the first forty-eight hours of the disease showed a death rate of only 13.1 per cent, whereas 157 pneumococcus pneumonias in the control series ran a death-rate of 26.7 per cent, practically twice as high.

TABLE I.—COMPARISON OF DEATH RATE IN TREATED AND CONTROL SERIES

Type.	Cases treated with antibody.			Control cases.		
	Cases.	Deaths.	Rate, per cent.	Cases.	Deaths.	Rate, per cent
Pneumococcus I . . .	157	21	13.4	175	41	23.4
Pneumococcus II . . .	78	22	28.2	76	31	40.7
Pneumococcus III . . .	57	20	35.0	60	24	40.0
Pneumococcus IV . . .	109	17	15.6	137	31	22.8
Total	401	80	19.9	448	127	28.3
Strep. hemolyticus . . .	21	10	47.6	21	9	42.8
Strep. viridans	7	1	14.2	10	1	10.0

TABLE II.—DEATH RATES FOR PNEUMOCOCCUS PNEUMONIAS RECEIVING ANTIBODY ON OR BEFORE THIRD DAY OF DISEASE. CONTROL SHOWS DEATH RATE FOR PNEUMOCOCCUS PNEUMONIAS ADMITTED TO CONTROL WARDS ON OR BEFORE THIRD DAY OF DISEASE

Type.	Cases treated with antibody.			Control cases.		
	Cases.	Deaths.	Rate, per cent.	Cases.	Deaths.	Rate. per cent.
Pneumococcus I . . .	56	5	8.9	68	16	23.5
Pneumococcus II . . .	24	5	20.8	25	8	32.0
Pneumococcus III . . .	10	1	10.0	19	7	36.8
Pneumococcus IV . . .	24	4	16.6	45	11	24.4
Total	114	15	13.1	157	42	26.7

It is difficult to explain why Type IV pneumonia should be favorably influenced by pneumococcus antibody solution, as this agent is not supposed to contain any immune substance against Type IV pneumococcus. Evidence is accumulating, however, to show that Pneumococcus Type IV is made up in part of atypical fixed types; in other words, many Type IV strains represent aberrant forms of Types I, II and III. If this hypothesis is correct, it would readily explain the beneficial effect of antibody on Type IV pneumonia.

Upon the evidence which has been presented we must conclude that pneumococcus antibody solution is a therapeutic agent of considerable power. The effect is most striking when the antibody is administered early in the disease, but it may also be of value in the later stage by neutralizing the circulating toxins. The most dramatic effects are observed in Type I infections, but good results can also be obtained in the other types when antibody is used early and in large doses.

More recently we have been studying the effect of antibody when administered subcutaneously. By this procedure, one avoids entirely the severe reactions which are often very uncomfortable and, in certain cases, actually dangerous to the patient.

This preparation is still a new remedy and will certainly be subjected to improvements as time goes on. A method must be devised for still further concentrating the antibody, and it would also be desirable to eliminate the shock reaction which follows intravenous injection. Even in its present form, however, pneumococcus antibody solution marks an important step in advance in the specific treatment of pneumonia, and I believe that it will be along this line that an efficient mode of treating the disease will ultimately be worked out.

MARCH 26, 1923

Quantitative Determination of Trypsin and Lipase in Gastric Contents

BY C. W. LUEDERS, M.D., AND OLAF BERGEIM, M.S., PH.D.

(Abstract)

Simple methods are described for the quantitative determination of trypsin and lipase in gastric contents. The methods follow closely those recently suggested by us (Lueders, Bergeim, Rehfuss: "Quantitative Determination of Enzyme Activity in Duodenal

Fluids," *Am. Jour. Med. Sci.* (in press), for determination of trypsin and lipase in duodenal fluids. They are modifications of the methods proposed by Gaultier (*Presse méd.*, 1919, xxxii, 115). They are titration methods and results are expressed in the number of cubic centimeters of $\frac{N}{10}$ acidity developed by the ferment action contained in 100 cc gastric juice following one hour's incubation at 37° C.

For trypsin, gelatin is used as a substrate and the amino groups liberated during digestion estimated by alkali titration after formaldehyde addition. Lipase is determined by its action upon an emulsion of olive oil, the acidity developed being titrated, following the solvent and liberating action of alcohol-ether upon fatty acids formed during digestion.

The principles of these methods are not new, but the authors believe that they have not previously been applied to gastric contents. They may prove capable of throwing considerable light upon the phenomena of duodenal regurgitation. Regurgitation appears to be a regulatory mechanism, interference with which, from the standpoint of either excess or deficiency, may be of clinical significance. Illustrations of trypsin, lipase, total acid and free HCl curves from fractional specimens are included.

Bronchoscopy in the Diagnosis and Treatment of Lung Suppuration[1]

BY LOUIS H. CLERF, M.D.
PHILADELPHIA, PA.

(*Abstract*)

Diagnostic bronchoscopy is indicated in all cases of lung suppuration. It is usually inadvisable in the presence of aneurysm, advanced arteriosclerosis and serious heart disease unless a foreign body is suspected.

[1] From the Bronchoscopic Clinic, Jefferson Hospital, Philadelphia.

The indications for bronchoscopic treatment in lung suppuration cannot be clearly set forth. Each case must be considered on its own merits in consultation with the internist, roentgenologist and surgeon. In a general way bronchoscopic treatment is indicated in the following:

Suppuration resulting from recent aspiration of a foreign substance.

Suppuration occurring as a complication of the infective diseases.

Suppuration with interference of drainage by some form of intrabronchial obstruction.

In addition, there is a great group of suppuration cases of long duration which is very often greatly benefited and not infrequently cured by bronchoscopic treatment.

It must be remembered that the bronchoscopist should always work in close harmony with the internist and the surgeon, so that all conditions arising will be carefully considered, indications met and the best interests of the patient served.

Lipodystrophia Progressiva: A Report of Two Cases, One of Which Showed Improvement Under Medical Treatment

BY THOMAS P. SPRUNT, M.D.

(*Abstract*)

About 70 cases of this interesting condition have been recorded since the first described by Barraquer in 1905. It is characterized by a remarkably complete disappearance of the subcutaneous fat of the face, of the neck and usually of the arms and upper portion of the trunk. This emaciation begins, as a rule, in childhood, first involves the face and then the lower parts progressively from above downward. Simultaneously, or beginning a few years later, there is an excessive deposit of subcutaneous fat on the

lower portion of the trunk, about the gluteal regions and on the legs, which may be in grotesque contrast with the emaciation above the waist.

The patients complain of a variety of psychoneurotic symptoms, and especially of the disfigurement, which is all the more important since almost all reported cases have been in girls or women. The other soft parts and the bones are not involved.

The 2 cases here described were typical, with the excessive emaciation above the waist and a well-nourished condition below the waist. They each showed, in addition, a marked psychoneurotic state and evidence of thyroid disturbance. In the first case there was a small, nodular thyroid struma, sight eye signs, sensitiveness to epinephrin, sensitiveness to heat, accelerated basal metabolism, normal pulse rate and no tremor.

The first case was under our direct observation for several weeks at two different intervals and has been in correspondence with us for several years. One of the generally accepted ideas hitherto held regarding this malady was that the emaciation is progressive to a complete loss of subcutaneous fat in the parts affected, and that once lost in a part the subcutaneous fat never returns; and, further, that attempts to fatten such patients result in an increase in fat of the lower part of the body alone, thus adding to the patient's deformity. The chief contribution of this report is, in my opinion, the demonstration that the factors producing the atrophy do not necessarily remain completely effective and that fat can be deposited again in the parts from which it has disappeared.

This patient was treated by rest and general upbuilding measures, and, in about four months, gained twenty-five pounds, attaining her calculated ideal weight. The weight was kept at this point during the next two years, and comparative measurements indicate that there was a redistribution of the fat, a part of which was deposited in the previously emaciated portions of the body. In addition to the general measures of rest, full diet, general massage, appropriate tonics, exercises, fresh air, etc., 3 grains of the extract of the whole gland pituitary were given after each

meal for about one year. What part this factor played, if any, in the resulting changes we do not know.

The second case did not remain under observation long enough for treatment.

The etiology of lipodystrophia is obscure. In general three different hypotheses have been suggested in discussions concerning the pathogenesis of this condition:

1. A disease of the nervous system, a trophic change, a trophoneurosis.
2. An endocrine disturbance.
3. A disease of the fat tissue itself.

Only three autopsies have been recorded, and these have been disappointingly incomplete and inconclusive. No gross change was discovered in the nervous system. In one case there was a cyst of the pituitary gland. In one case there was persistence of the thymus, in another complete atrophy of the thymus in a younger person than in the former case. In these autopsy cases, aside from some colloid change in the thyroid gland, there were no other striking endocrine features. Many of the cases reported from a clinical standpoint showed endocrine defects, but in such variety and in such contrast that an etiological relationship seems most unlikely.

The thyroid gland has recently been resected in the first case here reported, and it is said that the functional nervous symptoms have been much ameliorated. We are interested, of course, in observing any possible direct effects of this procedure upon the lipodystrophy. Fat injections of the subcutaneous tissues of the face have given satisfactory immediate cosmetic results, but they are soon absorbed. Fat transplantations have been suggested, but as far as we know have not been carried out in this disease.

Associated trophic lesions are rare.

One can draw no definite conclusions as to the etiology or pathogenesis of the lipodystrophy from the data available at present. It may be stated, rather unsatisfactorily, that these patients are the victims of a constitutional anomaly which first appears several years after birth, which may have as exciting

causes a trauma, a severe fright or an infectious disease, and which is frequently associated with other constitutional defects, especially in the endocrine system. The nervous symptoms are probably largely functional, and are due to the effect of the distressing situation upon a nervous system which may also have had a share in the constitutional weakness.

I wish to express my appreciation to Mr. O. C. E. Hansen, of the Johns Hopkins Medical School, for his courteous assistance in translation of Italian literature.

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Surgical Significance of Abdominal Pain

BY ARTHUR E. BILLINGS, M.D.

Of the many and varied surgical conditions which we encounter in the abdomen, pain is the most important symptom, and the one which more often influences the diagnosis and procedure than

any other. To correctly interpret its meaning as a symptom, one must have a fairly intimate knowledge of the rather complex nerve supply of the entire abdomen and its contained viscera before its significance is fully understood. It is aside from the subject for discussion to go into the detailed anatomy and physiology of the nerve supply of the abdomen at this time. However, I feel that it will not be out of place to refer to the main sources of innervation.

The abdominal parietes, front and sides, are supplied by the lower five or six intercostal spinal nerves, which give off anterior and posterior branches and terminate by entering the substance of the rectus muscle on each side and giving sensation to the mid-front of the abdomen. The hypogastric region and lateral flanks are supplied by the last dorsal and branches of the iliohypogastric and ilioinguinal nerves. It is interesting to note that the posterior wall of the abdomen receives its supply from the lumbar and sacral spinal nerves and not the dorsal. These branches are intimately connected with the lumbar and sacral plexuses, which lie on the posterior wall of the abdomen and are in close relationship with many of the viscera which are the frequent site of disease, and of which, when inflamed, pain is often the most conspicuous symptom. In addition to this, we have several large main nerve trunks entering the abdominal cavity which are closely connected with the other large distributing centers. The pneumogastrics, on each side of the esophagus, go to supply the stomach—the right to the posterior wall of the stomach and entering into the formation of the solar plexis, and the left to the anterior wall. The sympathetic enters the abdomen as two large trunks beneath the pillars of the diaphragm; then pass into the pelvis, ending in the coccygeal ganglia, having entered into the formation of several small ganglia (four lumbar, four sacral), which are the connecting stations between the various large sympathetic abdominal plexuses on the inner side and the spinal nerves externally. There are three other important trunks, on each side, passing from the thorax into the abdomen, viz., the great, the small and smallest splanchnics, which go to make up the semilunar ganglia and renal plexuses.

The phrenics, two other spinal nerves, descending principally from the fourth cervical, through their anastomatic branches communicate with the solar and hepatic plexuses and in this way have visceral connections, but, strictly speaking, do not enter the abdomen but supply the diaphragm, a not infrequent seat of baffling pain from the standpoint of diagnosis.

Next in consideration is that intricate interlacing of nerve fibers of the sympathetic system lying immediately upon the aorta, and at two points they are much more closely aggregated and constitute the great solar and hypogastric plexuses. The epigastric or solar plexus is situated behind the stomach and in front of the aorta and crura of the diaphragm. It receives the great and small splanchnics and the termination of the right pneumogastric, and supplies all of the viscera of the abdominal cavity from its ten distributing centers or lesser plexus.

The hypogastric plexus, located in front of the fifth lumbar vertebra, occupies the same relative importance to the pelvis and its organs as does the solar to the abdominal viscera. It will be observed that the nerve supply of the viscera and visceral peritoneum is derived from the various sympathetic plexuses, while that of the parietal peritoneum is derived directly from the spinal nerves (dorsal, lumbar, sacral and phrenics). The spinal nerves contain many more sensory fibers than do the sympathetic trunks. It is a long-established clinical fact that the parietal peritoneum is much more sensitive to manipulation and the traumatism of surgery under local anesthesia than is the visceral peritoneum or, in fact, any of the visceræ. This fact must also be remembered from a diagnostic point of view.

Before going further, it is necessary to refer to the connection that exists between the visceral nerve supply and that of certain well-defined areas on the body surface supplied by the spinal nerves and related through the same spinal segment. The brilliant work of Head, Ross, MacKenzie and others, but Head in particular, has resulted in establishing the exact relationship between sharply defined cutaneous tracts and viscera receiving their nerve supply through the sympathetic, but from the same spinal segment as

that received by the skin area. I quote from Head ("On Disturbances of Sensation, with Especial References to the Pain of Visceral Disease"): "Pain was in many cases associated with definite cutaneous tenderness. Moreover, the cutaneous tenderness was in many cases not confined to small spots or areas, but occupied whole tracts of skin with definite tenderness. I was then led to investigate the pain and accompanying tenderness consequent on disturbances of other organs, and I found that these sensory disturbances also followed definite lines. After Ross's most suggestive papers it seemed exceedingly probable that these areas bore some definite relation to nerve distribution, and I then began to investigate the distribution of herpes zoster in the hope that a skin lesion which was notoriously of nervous origin might throw some light on the meaning and significance of tender areas in visceral disease. I next attempted to determine to what level of the nervous system these areas belonged with the help of cases in which gross organic lesions were present. By this means it became apparent that each of these areas represented the distribution of a single nerve root or of a single segment in the spinal cord. Thus I was enabled to map out the areas supplied by the various segments of the cord on the surface of the body. It then became apparent that certain of these areas were never affected in visceral disease, and this led me to examine the sensory supply of the viscera from the sympathetic system. Now, Ross had already suggested that, in visceral disturbances, pain (and therefore, in my cases, tenderness) was referred along the distribution of the somatic nerves which come off from the same part of the cord as the sensory sympathetic fibers to the organ affected. Thus, if I could map out the somatic areas along which pain was referred in visceral disease, I could say, on Ross's hypothesis, what was the sensory supply from the sympathetic of the particular organ affected. By this means I obtained another scheme showing the distribution of the sensory sympathetic fibers analogous to that which Gaskill constructed for the motor and inhibitory fibers of the same system."

It is interesting to note that in this surface topography the

cutaneous zones correspond more closely to segmental supply than to individual nerve supply; also, the cutaneous zones are sharply defined and do not overlap as in the ordinary peripheral sensory supply to the skin. This most interesting work of Head's explains referred surface pain in visceral disease and enables us to follow it in many vague conditions to the affected viscera. Under normal conditions, pain in the skin is located with accuracy at the point receiving the stimulant, as, for instance, a pin-prick on any part of the body surface. This, to a large degree, is attributed by Howell to the temperature and pressure sense, and, when these are lost or destroyed in the skin surface, pain is then very inaccurately located. "Pain arising in the internal organs, on the contrary, is located very inaccurately, but is often referred to points on the skin and may be accompanied by areas of tenderness in the skin." Pain of this kind, when misreferred to the surface of the body, is designated as reflected pain. The explanation offered for this (Howell) misreference is, that the pain is referred to the skin region that is supplied by the spinal segment from which the organ in question receives its sensory fibers, the misreference being due to a diffusion in the nerve centers. As Head expresses it, "When a painful stimulus is applied to a part of low sensibility in close central connection with a part of much greater sensibility, the pain produced is felt in the part of higher sensibility rather than in the part of lower sensibility to which the stimulus was actually applied."

It is interesting that affections of the serous cavities, *e. g.*, the peritoneum, do not cause reflected pains or cutaneous tenderness as in the case of the viscera. Another important fact in referred pain is its occasional occurrence in the symmetrical area on the opposite side of the body. This is sometimes seen in renal disease, ovarian lesions, tubal pregnancy and breast inflammation, and is explained on the basis that depression of the cutaneous sense in a given area, with application of the stimulus to that point, will cause pain projection to the symmetrical area of the opposite side; if this is also involved, it may be referred to the area next above or below in the spinal order. On more than one occasion we have

found a stone in the opposite kidney, or a rupture of the opposite tube in ectopic pregnancy, to what was expected from the patient's localization of pain. The stimuli necessary to excite pain varies in different parts; for instance, those for visceral pain are not of the ordinary thermal or tactile types to which skin and mucous surfaces respond so promptly, but is believed by most investigators to be entirely due to tension in some form or other. Meltzer has attempted to explain all forms of colic on the basis of what he terms "the law of contrary innervation," which in the normal physiological peristalsis of the intestine is fulfilled in the simultaneous relaxation that always occurs just below the contracting segment. When this simultaneous contraction and relaxation are interfered with by the presence of inflammation, new growth or other disturbing lesions, pain is the result. The contraction of unstriped muscle is the most frequent cause of abdominal pain, and it is always referred along the midline of the abdomen somewhere from the ensiform cartilage to the pelvis. This is the reference segment for all of the hollow viscera, esophagus, stomach, intestine, and includes the bladder, uterus and bile ducts, while renal colic is distinctly unilateral in its reference.

When we come to consider the various surgical conditions of the abdomen which may be the cause of pain, we can only allude to the most important in the general way in the time at our disposal. Some of the lesions are chronic, others subacute and many are acute, depending upon the degree of severity and duration. Many acute conditions are but sudden and severe exacerbations of preexisting disease. It has been estimated that 90 per cent of all disease begins with or is accompanied by pain some time during its course. "Alone, pain indicates danger in general; in combination with other signs, it indicates danger in particular and guides the surgeon's hand to its source" (Richardson). In many cases it is the most important symptom; in others it is only an incident. Its location, the manner of onset, severity, duration, radiation, the relation it may have to certain events, like the taking of food, micturition, defecation, etc., demand careful consideration in their interpretation. It may be worse at night. Its relation to other symptoms,

such as vomiting, etc., must be given due thought. For certain lesions the severity, character and location may be quite typical. Age, racial characteristics and sex are entitled to consideration. Women, as a rule, endure pain better than men because of lessened sensibility to painful stimuli, and the phlegmatic are more uncomplaining than those with a highly developed nervous system. It is a well-established fact that in the aged there is occasionally an absence of pain in many of the lesions, such as appendiceal and gall-bladder inflammations, which are ordinarily productive of a great deal of pain. This is explained on the basis that in senility the sensibility to painful stimuli is lost to a great degree and sometimes completely. In the obese patient, tenderness and rigidity may be diminished or absent from anesthesia of the peritoneum as a result of fat deposit about the somatic nerve endings in the subserous space. The patient's interpretation of the character of pain may be of considerable value in arriving at the cause.

Pain ought always to be regarded from two aspects: In the one, the untouched patient manifests it; and in the other, the clinician elicits it. In the latter it is pain on pressure or tenderness which is always located about the seat of the lesion. Crile states: "Pain, like other phenomena, was probably evolved for a particular purpose; surely for the good of the individual. Like fear and worry, it frequently is injurious. What, then, may be its purpose? We postulate that pain is one of the phenomena which results from a stimulation to motor action." He also maintains that the only types of infection that are associated with pain are those in which the fixation of parts by continued muscular rigidity is an advantage that the type of infection that may cause muscular action when it attacks one region of the body may cause no such action when it attacks another. A striking instance of the protective action of pain is seen in acute abdominal infections, such as peritonitis, where there is segmental rigidity of the recti muscles in localized infections or generalized rigidity in diffuse infections. Peristalsis is almost absent; food is refused or may be ejected; the dorsal position is assumed and is not often changed. Muscular action has an adaptive and protective purpose in these infections.

The perforating lesions of the stomach, duodenum, appendix and, rarely, the gall-bladder cause that type of pain which should not be interpreted as meaning anything but the gravest sort of intraäbdominal lesion—the sudden onset, the terrific and unabating pain with boardlike fixation of the abdominal muscles is characteristic. The pain of acute pancreatitis and mesenteric thrombosis is equally severe and may not be relieved by heroic doses of morphine. In these lesions life is at once jeopardized by the intense suffering. In pancreatitis the pain is confined to the upper abdomen and nearly always passes with severity to the back. It is agonizing beyond endurance and frequently is the cause of collapse. In the gastric and duodenal perforations the history of the patient's condition previous to acute onset will, in a majority of cases, suggest quite accurately the nature and location of the lesion. This is particularly true of duodenal ulcer, where pain comes on one, two or three hours after taking of food, is gnawing or burning in character, is relieved by taking more food or alkalies for a time and is often described as hunger pain. The distinct periodicity to the pain and symptoms is another characteristic. In gastric ulcer pain is less characteristic, as a rule, and is generally aggravated by taking food. It is burning or scalding in character, is usually felt about the ensiform cartilage and is frequently referred to the region of the twelfth dorsal vertebræ.

In gall-bladder inflammation the pain is never so severe as that of the perforating lesions mentioned. A frank perforation of the gall-bladder with sudden soiling of the whole peritoneal cavity, as is seen in gastric or duodenal perforation, rarely occurs. When perforation takes place, it is usually walled off, and because of its anatomic position, results in a localized inflammation. Pain is not so severe and is localized, as is tenderness. In gall-bladder disease the character of pain may be variable. It may be that of an acute inflammation, the result of severe infection, which is accompanied by abdominal rigidity, tenderness and the other signs of acute inflammation; or it may be the pain of tension due to distention of the gall-bladder, chronic infection and the presence of stones. Tenderness in these cases can nearly always be

elicited by palpation over the gall-bladder. Referred pain may exist with either of the above conditions. The pain of gall-stone colic is usually intense, recurring in violent paroxysms and is referred over the hepatic and epigastric regions, and usually radiates to the back or the right shoulder. Vomiting, with profound nausea, is nearly always an accompanying symptom. Rarely the pain is referred to the left side and back.

The pain of peritonitis is sharp, stabbing or lancinating in character. Patients frequently described it as having "doubled them up." There is, of course, generalized tenderness and muscular rigidity over the involved peritoneum, and usually a point of maximum tenderness can be elicited about the site of the primary inflammation. Pain resulting from inflammation of the vermiform appendix is usually abrupt in onset. It is of varying degree in severity. It is usually referred to the epigastrium or around the umbilicus, and after the lapse of a few hours usually becomes distinctly worse in the right iliac fossa. The sequence of pain to other events is of great importance in the diagnosis of appendicitis. Pain is always the first symptom, and if pain is preceded by vomiting you can almost certainly exclude disease of the appendix. The pain is apt to be colicky in character until involvement of the peritoneum takes place, when its character may be somewhat changed. It may seem a little out of place to refer to purgation in these cases, but having seen so many diastrous results from it, I cannot refrain from mentioning it. It is unquestionably the cause of most of the cases of peritonitis that we see resulting from this disease. If these patients are not purged there is the same tendency for the inflammation to remain localized, even if perforation does occur, as is manifested in infections of the gall-bladder and pelvic appendages. Moynihan says that in appendicitis perforation spells purgation, and in his large experience he pens the sequence of events in pain, aperient and perforation. He further says, "I, therefore, do not hesitate to say that in almost every instance of acute peritonitis due to the perforation of the appendix it is the treatment directed to the relief of the condition that is the cause

of the serious and so often fatal catastrophe." He has expressed himself most clearly and forcibly in an amended nursery rhyme:

> Perforation means purgation,
> With the appendix kinked and bad;
> Both food and drink will worry him
> And aperients drive him mad.

Disease of the pelvic appendages in the female is a common source of pain. A bimanual examination should never be omitted, especially when a woman complains of backache. An ovarian cyst, a pus tube or a right ureteral stone is often diagnosed as appendicitis; and a lacerated cervix or perineum with a retroverted or prolapsed uterus treated for kidney disease or lumbago. Pain of these organs is often described as "bearing-down pain or heavy backache." Pain may also be reflected down the front of the thighs. The pain of ruptured ectopic pregnancy is usually severe in character but does not approach the intensity of the perforating lesions that we have described. The pain, of course, is more severe over the lower abdomen, and is occasionally located on the opposite side of the rupture. Patients sometimes describe it as "if something had given way or had burst within the body." The pain of intestinal obstruction is usually sudden in onset. It is violent in character, continuous, with fierce exacerbations. The continuous pains are due to the constriction of the intestine and the exacerbations to the colic of an overactive peristalsis.

The location of pain varies with the site of obstruction. It has been shown by MacKenzie that the pain of obstruction is always located along the midline of the abdomen, the zone for the small intestine embracing the umbilical region corresponding to the levels of the costal margin above and the crest of the ilium below, while that of the large intestine occupies the hypogastrium. One may be aided in the localization of this pain by remembering that it begins well above the point of obstruction, gradually passing lower and lower until it reaches a climax. If when the pain stops this part be noted, the situation of the obstruction can be localized within certain limits. Unfortunately the limits are still wide, nevertheless the information is extremely useful as an aid to diagnosis.

The pain of tuberculous peritonitis and localized tuberculous processes is not in any way characteristic, but is usually either sharp and stabbing in character, like that experienced in other infections of the peritoneum, or else it is colicky in type, due to interference with peristalsis or involvement of the intestinal tract.

There are many other less common surgical lesions of the abdomen, including ureteral calculi, intestinal tumors, mesenteric cysts, eroding and ruptured aneurysms and typhoid perforations, etc., which we will not take up.

In the examination of a patient for the purpose of eliciting pain or tenderness, one must nor forget the influence of position on pain in the various organs. The occurrence of a painful position points to a localized process. This is frequently found to exist even when the pain appears to be diffuse, as in acute appendicitis. Painful position may be present in cancer, gall-stones or stone in the pelvis of the kidney. The lateral position is frequently found to be painful, for it involves the most favorable conditions for abnormal displacement and traction if tumors or adhesions are present. The influence of motion is of considerable value in acute inflammatory and suppurative conditions of the abdomen, as when the patient anxiously maintains the dorsal position and carefully avoids turning to either side. A transmitted jar from walking or going down steps will frequently cause pain when there is present a pyonephrosis and appendiceal abscess or acute inflammation of the gall-bladder.

In the consideration of abdominal pain one must keep constantly in mind the many extraäbdominal conditions which so closely mimic many of the acute abdominal lesions. Among the most conspicuous of these are the referred pain of pneumonia, pleurisy, diaphragmatic infections, acute dilatation of the heart, gastric crisis of tabes dorsalis, acute coxitis of children, spondylitis, herpes zoster and angina pectoris. The abdominal symptoms of the latter condition are sometimes described as angina abdominus. Some of the more common non-surgical intraäbdominal conditions, such as lead-poisoning, acute gastritis, colitis, etc., may be excluded in the diagnosis of a surgical lesion by careful history taking and a proper correlation of symptoms with thorough physical examina-

tion. There are conditions in which a positive preoperative diagnosis cannot be made other than that of an acute abdominal catastrophe. In these cases, with the use of all diagnostic means at hand, accurate interpretation of history and symptoms and a proper display of surgical judgment, a timely operation may be performed or an operative casualty averted.

Someone has classified the causes of pain into (1) muscular, such as is seen in the passage of ureteral or biliary calculi; (2) that caused by irritants of a chemical or microbic nature, such as is seen in peritonitis from perforation of a gastric or duodenal ulcer or gangrenous appendix; (3) from undue distention of encapsulated organs, such as the liver, spleen, pancreas, kidneys, ovaries, etc. This distention may be due to abscess or cystic formation or the infiltration of new growth or inflammation; and lastly, where the source is found in the invading process of an aneurysm, where the pressure is from without, or there is erosion and exposure of nerve trunks.

When we remember that these complicated plexuses and network of nerve trunks, leading to and from the abdomen, conveying all kinds of nervous impulses, some motor, some sensory, many secretory and others vasomotor, and maintaining such an intricate relationship one with the other, and all with the whole nervous mechanism, it is well-nigh impossible not to believe that when any material disturbance is brought to bear upon a particular plexus, through undue stimulation of a part supplied by its nerves, that a much wider effect must be produced than would otherwise be the case if no such association existed. With such considerations in mind, it is possible to see some explanation of the many and varied phases which the same disease may present in different individuals. In one case motor derangement may feature the disturbance, in another it will be altered secretions; in still another, sensory or vasomotor symptoms will predominate.

I wish to make grateful acknowledgment to such authorities as Head, MacKenzie, Ross, Crile, Moynihan, Howell, Meltzer and Richardson, whose works I have used freely in the preparation of this paper.

APRIL 23, 1923

Endarteritis Obliterans (Luetic): Report of a Case

BY MITCHELL BERNSTEIN, M.D.

M. M., a Russian, aged fifty-three years, an ice man, complained of severe burning pain in the second toe of the right foot, commencing late in September, 1922. He also complained of shortness of breath.

His father and mother both died at seventy years of age. His mother had had four miscarriages and twelve children, all dying in youth excepting the patient, and one sister, who lived to thirty-three years of age.

He has been in America for twenty-four years; has been married for thirty-two years and his wife has had eight children, four of whom are living and well and four having died, one each of pneumonia, scarlet fever, influenza and an accidental death.

The patient denies Neisserian and luetic infection, but has had measles, chicken-pox, scarlet fever and typhus fever.

His present illness dates back to twelve years ago (1911), when he felt a burning sensation in the second toe of the left foot. This burning sensation continued, together with excruciating pain, for several weeks. The toe-nail was removed, the supposition being that an abscess was present. The symptoms continued for six months longer, when the distal portion of the toe sloughed off and healing began. This severe pain, resulting in the loss of the end of a toe, recurred once yearly for seven consecutive years, each attack lasting about three to five months.

Late in September, 1922, after a pause of four years, the second toe of the right foot became involved. Burning pain was continuous, the toe was swollen and purplish in color and hot to the touch. In November the skin became whitish and wrinkled over the distal portion. Ulceration and a line of demarcation encircled the distal joint. The loss of the nail and soft tissues of the end of the toe occurred in December, 1922, followed by gradual healing.

Relative to his dyspnea, the patient stated that in July, 1920, he was seized suddenly with an attack of shortness of breath and considerable precordial pain and flashes of darkness before his eyes. These attacks have been continuous, and are brought on by the slightest exertion.

A, end of toe still sloughing; B, horny remains of previously involved toes.

Physical Examination. He is a well-nourished man, having an anxious expression, the face being somewhat suffused. The pupils are equal and react to light and accommodation. The media are clear, disk is of good color and the margins well defined. The arteries are contracted and show evidences of sclerosis, but no hemorrhage or white spots. The nose is negative; the pharynx is injected. Many root stumps are present in the jaw, with nine teeth presenting evidence of pyorrhea. A visible carotid pulsation is evident in both sides of the neck, and there is slight fulness in the left supraclavicular fossa. The left side of the chest is bulged. The lungs are filled with piping rales. The apex beat of the heart is in the sixth left interspace at the anterior axillary line, and percussion dulness extends 5 cm. to the right of the midsternal line.

Impairment in the first interspace, both to the right and to the left for about 2 cm. from the sternum, is noted. On auscultation a diastolic aortic murmur is heard at the aortic cartilage and transmitted down the left side of the sternum. The temporal arteries are sclerotic, the brachials are tortuous and hard. The radials are sclerotic and a Corrigan's pulse is present. The femorals are palpable and hard. The popliteals cannot be made out with ease. The dorsalis pedis arteries are hard, but the pulse is present. The pulse is also present in the posterior tibial arteries, but is very weak. The veins in the arms are very prominent. Blanching of the feet is evident upon elevation and cyanosis is marked below the ankles when the feet are lowered. Slight edema below the ankles is present at times.

The knee-jerks are slightly diminished and Romberg's sign is negative. The feet present the most striking feature, in that the ends of all the toes, excepting the large toes, are missing, some being replaced by a horny, nail-like extension. The end of the second toe on the right foot is still in the process of sloughing, as seen in the accompanying illustration.

Laboratory Reports. The blood Wassermann is four-plus. The urine on several examinations shows traces of albumin, with the specific gravity varying from 1015 to 1022, 50 to 150 pus cells to the field, no blood; hyaline and finely granular and coarsely granular casts are present. Hemoglobin, 80 per cent; red blood cells, 4,200,000; white blood cells, 12,000; color index 9, polymorphonuclears, 71 per cent; small mononuclears, 21 per cent; large mononuclears, 4 per cent; transitionals, 1 per cent; eosinophiles, 3 per cent. The systolic blood-pressure varies from 140 to 160 mm. of Hg. and the diastolic ranges from 30 to 40 mm. of Hg.

Fluoroscopic examination of the chest reveals an enlarged heart and non-aneurysmal dilation of the aorta. The roentgen-ray study of the feet reveals a marked rarefaction of the bones. There has been absorption of the terminal phalanx of the fourth and fifth toes of the right foot, and probably beginning in the same toes of the left foot. There is calcification of the dorsalis

pedis artery, and possibly of some of the other vessels. There are distinct atheromatous arterial changes in the distribution of the artery along the internal border of the tibia. The heart is dilated and hypertrophied, and extends well to the left. General dilation of the aorta, non-aneurysmal.

Catalepsy in Epidemic Encephalitis: A Clinical and Pathological Report of a Case of the Cataleptic Type, with an Explanation of the Phenomenon[1]

BY A. E. BENNETT, M.D.

AND

JOHN H. MUSSER, JR., M.D.

(*Abstract*)

They reviewed the literature in which catatonia has been reported as a symptom in organic brain diseases and also the reported cases of epidemic encephalitis in which catalepsy has been present as a symptom.

Catalepsy is a preferred term in describing these cases, as the term catatonia implies the mental state seen in dementia precox, and the cases of encephalitis that have exhibited this phenomenon of maintaining fixed positions have not shown any other schizophrenic trends.

Two theories have been advanced to explain this amyostatic syndrome; hypermyotonia with rigidity and catalepsy seen in encephalitis: (1) That the condition is due to interference with cerebral function; (2) that the phenomenon (catalepsy) is part of the Parkinsonian rigidity of encephalitis and is the result of disease of the striatal or extrapyramidal system. This theory is the most probable and was shown pathologically in their case.

[1] From the Philadelphia General Hospital.

Case report of a sixteen-year-old colored girl, whose illness began with choreic movements, followed by profound lethargy and general muscular hypertonicity and catalepsy. She was ill three weeks and died of bronchopneumonia. At necropsy fairly well-localized lesions were found in the striate system (especially the thalamus and substantia nigra) to account for the cataleptic state.

They conclude in their paper that patients of this type that recover will show Parkinsonian sequelæ, that this is a severe type of disease, that muscular hypertonus in encephalitis is a frequent symptom and may reach a degree sufficient to enable the patient to maintain fixed attitudes or catalepsy. This hypermyotonia with rigidity should not be mistaken for a schizophrenic reaction and should easily be differentiated by neurological findings, spinal fluid examinations and the absence of other symptoms of dementia precox.

Chronic Arthritis, particularly, Rheumatoid Arthritis, and its Treatment by Injection of Coley's Toxins

BY ROBERT G. TORREY, M.D.

AND

THOMAS KLEIN, M.D.

(*Abstract*)

There is unfortunately much confusion in the consideration of chronic arthritis. Generalized multiple polyarthritis is classified under many different names, but can be in a general manner divided into two great classes, atrophic or rheumatoid arthritis and hypertrophic or osteoarthritis.

Rheumatoid arthritis is characterized by atrophy of muscles, joint structures and skin, and later by contractures and usually fibrosis. Atrophy of muscle groups is rapid. Atrophy contracture and fibrosis are the causes of disability in these cases. Bony ankylosis does not occur. In this group of cases the condition is progressive, with repeated exacerbations of acute inflammation

of the joints, in which the joint inflammation resembles the arthritis of rheumatic fever.

Rheumatic fever must be clearly differentiated, inasmuch as the arthritis of rheumatic fever is not progressive, does not tend to become chronic and does not cause joint destruction or permanent joint damage. This infection causes cardiac damage, and in the treatment of rheumatic fever the cardiac condition is the sole cause of concern.

In rheumatoid arthritis the toxic effect of a focal infection is usually concerned, but it seems probable that the distribution is determined by central nervous system control or influence. The perfect symmetry in development of the lesions, the symmetrical muscle atrophies and the behavior where trophic conditions are altered, as by hemiplegia, indicate the important participation of nervous influences.

Coley's toxin has been used in the treatment of these cases. By increasing doses a good effect on the inflammatory condition is secured. The severity of reaction to this toxin, given intramuscularly, is not uncertain, as in the case of intravenous protein therapy, and treatment can be continued over a long period of time. Focal infection must be sought for and eradicated. Contractures must be attacked by mechanical methods.

Practical Dietetics and the Modern Conceptions of Metabolism

BY EUGENE F. DU BOIS, M.D.
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(Abstract)

In the so-called "fluid diet" the chief constituent, milk, forms a large coagulum in the stomach. Many so-called solid foods become practically fluid in the stomach. Diets planned to give the patient a measured amount of fluid usually show an error of

about 100 per cent. Large amounts of water are contained in the so-called solid foods—*e. g.*, potato 78 per cent water, tomato 94 per cent water—as contrasted with milk which contains only 87 per cent water. The pure dried foodstuffs furnish considerable amounts of water when oxidized in the body:

100 grams of	fat	furnish	107	grams	water
" "	starch	"	55		"
" "	protein	"	41		"

The clinical measurement of the output of fluids usually contains a similar error of about 100 per cent if we neglect the water of the feces and the average of 700 cc of water excreted daily through the skin and lungs.

A protein-free diet in nephritis does not cause a protein-free metabolism, and men never metabolize less than 15 to 20 grams of protein a day. If given in small, divided doses, this amount of protein in the diet does not increase protein metabolism.

The typhoid on a liberal diet metabolizes more carbohydrate and less fat and protein than when he is on a low diet. The total number of calories is not changed.

In diabetes the metabolism may be surprisingly independent of the food administered. Richardson, Mason and Ladd, using the respiration calorimeter, determined the exact combustion of protein, fat and carbohydrate when the diabetic patients were fasting. They planned diets containing exactly the same amounts of these foodstuffs and fed the patients every two hours. In this manner they supplied the patient's blood with exactly the same materials that he oxidized fasting. There was practically no change in the metabolism. They then tried double the amount of fat in the diet, but there was no increase in the fat metabolism, and the excess was stored in the body. This shows that the fatty acid-glucose ratio of the food may bear no relationship to the ratio of these substances actually oxidized.

The food that the patient metabolized is seldom the same as that which the doctor orders in the diet.

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Diseases Due to Deficiency in A-vitamines

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(*Summary*)

Blindness in children has decreased all over the world, principally because of precautionary measures taken against ophthalmoblenorrhea. In Denmark, however, xerophthalmia has caused an increase in the number of blind children. This disease was first described in the middle of the last century by German ophthalmologists in wasting diseases, especially in badly nourished or debilitated children. "Epidemics" of this disease were later described in various countries, and primitive medicine has from ancient times treated this disease with fats, especially cod-liver oil and liver. The disease was considered rare in Europe. Since the opening of the pediatric department of the Danish State Hospital, in 1911, an increasing number of such cases were admitted, so that the author had opportunity to study the disease, about which he published various communications between 1914 and 1922.

These investigations showed that the disease was due to a lack of some particular substance, which was present in fresh milk but not in separated milk or buttermilk. The substance in question, therefore, was associated with the butter-fat and was also found in abundant quantities in cod-liver oil. Lack of fat, as such, was not the cause, since xerophthalmia occurred in children receiving margarine or pork-fat.

Owing to the animal experiments of Osborne and Mendel, and particularly of McCollum and his collaborators, we now know that the cause of xerophthalmia is a lack of the fat-soluble A-factor. This substance, which is notably found in butter-fat and cod-liver oil, must be constantly supplied to the growing organism in order that growth may take place. A long deprivation leads to xerophthalmia and death.

Infants deprived of milk, cream and butter will develop xerophthalmia. Vegetables, fruit and green-stuff given now and again will only help a little, as a small child can only digest trifling amounts of green-stuff. Oxidation and repeated heating destroys the A-factor in milk.

The first symptoms are inhibition of growth and depressed psychic and bodily vitality.

An increased susceptibility to infections is present. Later night-blindness and xerosis (dryness) of the ocular conjunctivæ occur, followed by dryness and necrosis of the cornea and inflammation. Blindness ensues if the disease is not recognized and treated in time. When the cornea is affected the lesion heals with hazy and opaque scars.

Children receiving no milk-products whatever become atrophic, the intestinal trouble then dominating the clinical picture; in this case several other factors besides the fat-soluble A are missing.

If only a little butter-fat is given in addition to the carbohydrate diet, edema may occur, but not xerophthalmia or increased susceptibility to infections.

In bigger children a lack of the A-factor may merely cause inhibition of growth and infectious diseases of various types. This was seen during the war in Germany, where the infants, however, were not affected, since they received regular rations of milk and milk-products.

In Denmark 600 to 700 cases are known to have occurred between the years 1909 to 1920.

The towering prices on the world market for dairy-products caused these to be pretty nearly inaccessible for the poorer part of the population, while separated milk or buttermilk could be had at a very low price. Consequently there occurred a steady rise in the number of cases up to the beginning of the unrestricted submarine warfare, which stopped the import of margarine and the export of butter, so that every individual was entitled to a ration of butter. The number of xerophthalmia cases at once declined to a small minimum and has remained low ever since, especially in its severer forms.

The disease is treated with fresh milk, cream, butter or cod-liver oil. Xerophthalmia first disappears, then the child begins to thrive and gradually throws off the infections.

The fat-soluble A-factor plays a role in establishing immunity. Large quantities are used up during infections and also during growth.

It is the child — the growing organism — that is afflicted by xerophthalmia, and also that the largest number of cases are observed in the spring, at which season the most remarked growth takes place.

This seasonal incidence has been ascribed to a difference in the A-factor content in the milk of stall-fed and grass-fed cows. It is difficult to believe that the milk of a healthy cow giving abundant milk can be deficient in A-substance. This has not been proved, and the experiences with xerophthalmia in Denmark are against this supposition.

Finally, the author wants to mention that xerophthalmia and rickets are only very rarely seen together in the same child. Rickets can therefore hardly be due to the absence of fat-soluble A in the food or in the organism.

DISCUSSION

Dr. Edward B. Meigs: I want to begin by expressing my deep sense of gratitude to Dr. Bloch for the very interesting work in a general way and also for bringing such definite and incontrovertible evidence to show that the fat-soluble vitamine is important for human beings. Perhaps I ought to admit at the outset that I am an enthusiast on the subject of the causation of disease by dietary deficiencies. I believe we are perhaps not just at the beginning, but well started in a movement in medicine and physiology which will probably ultimately be as important as the development of bacteriology under the leadership of Pasteur and others, and which will furnish the proper supplement to this development in the way of showing that we must not look entirely to chance infection as an explanation of disease. It is becoming more and more evident that there is a large class of diseases with which infections have nothing to do at all, and again, that between these diseases and the more purely infectious diseases, that is the diseases to which all human beings are subject irrespective of their nutritive condition, there is a large

intermediate class in which susceptibility to bacterial infection is increased by the dietary state. It is, of course, necessary in this field that we should go a good deal into detail. We know that some animals are affected by the lack of certain dietary essentials and that others are not. It is, of course, notorious that rats—and it is becoming pretty well known now that cattle also—are almost insusceptible to scurvy, whereas guinea-pigs and monkeys are highly susceptible. It is therefore necessary in each case with dietary deficiency that we should have definite evidence that the particular animal which we are most closely interested in—in this case, I am sure, the human being—is affected by the particular deficiency in question.

Dr. Bloch's paper is so very complete that as he went along practically all the questions that formed themselves in my mind in the early part of it were answered by what he said later on. It occurs to me as being a very interesting aspect of his results in view of the fact that there has been so much controversy as to whether xerophthalmia and rickets were caused by deficiency of the same or of different dietetic factors, that his observations add one strong piece of evidence to that which is already on hand to show that in the A factor of McCollum and the D factor of McCollum we really have two different entities. One question which did occur to me was the explanation for the fact that the incidence of xerophthalmia began before the war. As I remember Dr. Bloch's chart, the frequency of the disease began to go up about 1909, and the rise at this time was quite large in comparison to the subsequent further rise in 1914–15, when the war began. Of course, it may be that I missed the explanation of this, but I do not understand just what it was, if Dr. Bloch gave it. The other point that I would like to be clear on is the question of the value of milk from different sources in preventing xerophthalmia. This, again, is a point which touches me very closely on account of my own particular occupations, and I have rather thought that there is pretty strong evidence to show that milk does vary very considerably both in its power to prevent rickets and in its power to prevent scurvy, according to the way the cows are fed. On the subject of rickets we have the work of Shick and the English Commission among the children in Vienna, where they found, apparently, that there was a difference in the power of milk to prevent rickets according as the cows had come in from the pastures or had been for a long time on winter feeds in the stall. On the question of the butter vitamine, more especially, although here again we are perhaps a little mixed up between the two, we have had a paper which I think Dr. Bloch may have heard at the recent meeting of the Dairy Congress by Captain Golding, of the Reading Station in England, in which he found that the power of milk to produce growth in rats (and I think Dr. Bloch

found its power to prevent xerophthalmia also) was highly dependent upon whether the cows were out on pasture or winter fed. Captain Golding also made the highly interesting observation that the potency of milk from stall-fed cows for producing growth in rats was very much increased by feeding cod-liver oil to the cows. I am not quite sure whether I understood Dr. Bloch on this, but I understood him to say that he thought the evidence from his work on human xerophthalmia was rather opposed to this point of view, and I would like him to make it absolutely clear just what his view is.

Dr. J. Claxton Gittings: About two years ago I attended a hearing before the Senate Committee on the Filled Milk bill in Washington when Dr. McCollum presented his testimony in regard to the production of xerophthalmia in rats as the result of deprivation of the A factor. The Senator conducting the hearing was anxious to find out whether we had actually seen these cases in human beings in this country and it was impossible to prove that there has been any considerable occurrence of them. I could see plainly that the Senate Committee was somewhat dubious as to the practical importance of the A factor in cow's milk in preventing xerophthalmia. I wish they could have seen Dr. Bloch's charts, and they certainly would not have been in doubt. Has Dr. Bloch a definite idea as to the amount of cod-liver oil which would prevent the development of this disease? I ask that in connection with the rather widespread use of the infant food called S. M. A., in which the fat of milk is replaced by vegetable and animal fat bolstered up with cod-liver oil. These vegetable oils which comprise the bulk of the fat are largely devoid of the A factor. In 1 liter of the prepared food the actual amount of cod-liver oil would not exceed 4 or 5 cc. If the other fats are deficient in the A factor it is important to know how much cod-liver oil can be relied upon to prevent the development of xerophthalmia.

Dr. D. J. McCarthy: I would like to ask Dr. Bloch a question in reference to the importance of bacterial invasion in the dissemination of diseases like xerophthalmia and scurvy. In the Mourman district in Russia in 1916 and 1917, where many of these cases of scurvy occurred, there was a definite feeling on the part of Russian Sanitary Corps officers that a bacterial invasion was responsible for the precipitation of the scurvy epidemic, if not the scurvy itself. A good deal of evidence was found to support the contention, some of the evidence being that in military units or certain divisions scurvy developed; where these units were isolated the scurvy did not develop in the rest of that division. There was a very large amount of scurvy, as I said, leading to the theory on the

part of some of the Sanitary Corps that it was purely a bacterial disease. I think the general feeling was there as here that scurvy was a vitamine disease, but that the precipitation of the scurvy was due to a definite type of bacterial invasion.

This led to one interesting point of view: that is, they declined to give typhoid and paratyphoid in the spring months for the reason that they said this had a tendency to precipitate scurvy and diseases of this kind. I would like to hear from Dr. Bloch whether he considers that bacterial infection on a deficient vitamine finding is a factor in scurvy epidemics. I think the section is very deeply indebted to Dr. Bloch for the very excellent presentation of an interesting subject.

Dr. C. B. Farr: As I was late I do not know whether Dr. Bloch said anything about the influence of light, such as has been used in rickets, in the treatment of xerophthalmia. Practically it is found that the use of light in rickets, and particularly of the violet rays, will more or less take the place of vitamines. I wonder if this might also be true of xerophthalmia?

Dr. Bloch (closing): You must first excuse me, but it is twenty-five years since I was in England and therefore I cannot speak English as well as I ought to. Therefore, I am unable to answer all the questions. I could answer them if I spoke English better. First, as to the frequency of xerophthalmia in Denmark before the war, I am sure we have had xerophthalmia for many years and all of the cases I received in my clinic were formerly treated as infectious eye diseases. We treated them with zinc. It is nearly impossible to distinguish this picture from that of gonorrheal ophthalmia. I am equally sure you will find it all over the world. It was only after my investigations that I recognized these eye lesions as this special disease, xerophthalmia. That it increased during the war was due to the fact that we had a scarcity of milk and butter in Denmark during the war. All the milk and butter was going to England and the poor people could only get skim milk. We can get skim milk in Denmark for next to nothing. I know, of course, the expedition because they sent me their report and they have translated my Danish investigations on xerophthalmia several years after I published them in Danish. If you look in the reports from the B. A. you will see about xerophthalmia. They have not found xerophthalmia in Germany, or only a very few cases. You were saying that the milk from cows fed in certain ways contained less A vitamine and used this as an explanation of the seasonal incidence of xerophthalmia. In Denmark the cows are always during the whole summer on pasture, and during the winter they get hay, and in hay there is a great

amount of A vitamine. Therefore, it seems to me the milk in Denmark will always contain this substance. I do think that a healthy cow can produce a milk without vitamine. I cannot believe that a milch cow can produce any milk without getting A vitamine. It is not shown; the animal experiments show nothing. I do not believe that you can make the milk just as you will by different feeding. When a cow does not get enough A stuff from the food the milk production stops. As to the necessary amount of cod-liver oil: When a child with xerophthalmia receives 5 gm. a day the disease will disappear in eight days when not too far advanced. I think only a few grams a day is enough. When the last gentleman spoke about scurvy being due to an infective agent I do not think this is so. I think it is proven that scurvy is due to lack of a certain substance in food. You know that it was a Scandinavian, Axel Holst, in Christiania, who first produced experimental scurvy in pediatrics, and I think it is proved that scurvy is due to lack of A vitamine.

The Rhinopharynx as a Site of Focal Infection

BY MYER SOLIS-COHEN, A.B., M.D.

(*Abstract*)

When a culture is made in a patient's whole coagulable blood, only those organisms grow up which are pathogenic for him. The organisms, even the predominating ones that grow up on ordinary culture, may be non-pathogenic to the patient harboring them, and hence not the cause of any infection present. In infection of the upper respiratory tract the infecting organisms are seldom confined to the tonsils and nasal accessory sinuses, but are usually present on the rhinopharynx and elsewhere, where they frequently remain after operative measures or administration of vaccine prepared in the usual manner, which may not contain the etiologic organism. Sometimes their chief focus is in the rhinopharynx.

The different portions of the upper respiratory tract vary in their bacterial flora, that of the rhinopharynx not necessarily

being identical with that of any other area. Organisms, pathogenic to an individual, persisting in the rhinopharynx may produce a variety of lesions in various parts of the body just as when present in any other focus.

DISCUSSION

Dr. Ross Hall Skillern: Dr. Cohen said the rhinopharynx is sometimes the chief focus for focal infection, and an exceedingly persistent one. I am going to take this as a text for my discussion and see just how the thing occurrs. Now, before we discuss the pathological end, let us review for a moment or two how the nasopharynx is protected from the onslaught of these bacteria. Both the nose, the nasopharynx and the sinuses are protected by ciliary action. The cilia move in certain ways, regarding the sinuses toward the ostium, in the nose they move faster and faster toward the rhinopharynx, thus having a tendency to force everything out of the sinuses and into the nasopharynx. That is No. 1, or the first natural reaction to infection. No. 2 is the glandular secretion which the nose and sinuses possess. While it may not have a distinctly bactericidal power, nevertheless it has an inhibitory power over all organisms. Regarding the work of Törne some twenty years ago: Törne procured the heads of freshly slaughtered calves and splitting in a certain way so as to open the maxillary sinus, applied lamp-black and noted that the fine particles of lamp-black moved toward the ostia at about the rate of 1 cm. a minute and backward into the nasopharynx. The action of the cilia decreased in a few minutes after the calves were dead. He also procured nasal secretion, and, using anthrax bacilli, found while it did not have a bactericidal action it inhibited the growth of these bacteria. So I think we can rule out what Dr. Cohen said regarding the nasopharynx and the occurrence of focal infection in distant portions of the body without some lesion being present in the nasopharynx. Now, what then are the causes of the nasopharynx becoming the seat of pathological activity? First of all we have a predisposing cause. If we have any cause which will prevent aëration and ventilation of the nasopharynx we will immediately have a good seat for the propagation of bacteria. What will cause it? Anything which will cause the obstruction of one nostril, such as nasal polyps or hypertrophy. That will only cause it on one side. An actual cause must be something which interferes directly with the action of these cilia, such as inflammatory conditions, congestion or adenoid tissue, in fact any pathological condition that will interfere with the action of the cilia, also swelling of any sort crowding down upon and interfering with action of alæ of the

nose. Now, then, when this occurs we do have an ideal place for the propagation of the infection—heat, moisture and quiet—and only when the cilia do become paralyzed or inhibited can we have this ideal place for this action. Dr. Cohen said the tonsils were found normal in many cases where bacteria were found in the nasopharynx, and many times previous to a year ago I found the tonsils normal up until we began to evert the tonsils and by means of platinum loop obtained cultures from deep into the crypts, so that cultures must be made properly from the tonsils in order to find the germ that happens to be present. As I take it, in the rhinopharynx, too, we must have some macroscopic lesion. I do not think the rhinopharynx can harbor these germs without any symptoms whatever. That is, however, entirely my own personal view. Dr. Cohen's theory will be well conceived when he can prove direct relation between the pathological lesion and the remote condition. That can only be proven, if, for instance, he can treat the nasopharynx alone with beneficient results to the condition that has obtained, then we will have to admit that such is the case. And so my opinion remains about the same. While the rhinopharynx may be the seat in certain infections which cause trouble farther on, we must regard it as a rather intermediate station, and in order to reach the true source we must go still farther into the depth of the sinuses and the crypts of the tonsils.

Dr. William F. Moore: I think that discussions of this sort are good for us all. I agree with Dr. Skillern that we should have some objective evidence of disease in the rhinopharynx if we are going to call it a source of infection. I am reminded of a case we saw in the Jefferson Hospital some months ago where the condition was later diagnosed as ethmoiditis. This child came from the shore without any direct evidence of involvement of the ethmoids and became entirely well while in the hospital. When he returned to the shore a second time he started to run a temperature and was again brought back to Philadelphia. On his second admission the child showed a very evident discharge of mucopus for the first time in the pharynx and was x-rayed, but at the first admission you would have said that this child had absolutely no evidence of the ethmoid infection which later proved to be the source of trouble. Now, regarding Dr. Cohen's tracheobronchial asthma, in which he said he has observed mixed infections from his cultures taken from the nasal pharynx: we have been doing a small amount of work in this line and find we can only depend on these cultures when they were taken directly from the parts themselves, namely, the bronchi or trachea, and we necessarily expected to get a mixed infection when the cultures were taken from the nasopharynx. Specimens taken from the pharynx are too liable to contamination from the aural and nasal cavities.

Dr. Cohen (in closing): The point of the matter is that laryngologists frequently fail to cure the secondary lesions in various parts of the body by the removal of diseased tissue from an infected focus. The patient has diseased tonsils removed; the heart disease continues. Why? Because I believe the bacterial infection has not been cured, and when you culture you find this is true. The organism that was in the tonsil that has been removed remains behind and lives in the rhinopharynx. Dr. Skillern says that the rhinopharynx has to have evidence of disease in order to have bacteria present. In cases in which organisms pathogenic for the patient were found in the upper respiratory tract laryngologists had made a thorough examination and found no abnormality present. The therapeutic test I did mention in some cases. When you have a patient showing signs of focal infection, arthritis and other lesions, and a vaccine made of organism found in the rhinopharynx produces a cure, that is suggestive evidence of the rhinopharynx having been the site of the focal infection. Dr. Skillern does not believe that organisms can remain in the rhinopharynx that is not diseased. The fluid bathing the mucous membrane will kill them. The fact is, however, that organisms do stay there and flourish. And, as I mentioned before, in a case of appendicitis the same organism is sometimes found both in the throat and in the appendix, or in the feces in a case of cholecystitis the same organism that the blood cannot kill will often be found both in the throat and in the duodenal washings.

A Method of Teaching Students the Diagnosis of Pulmonary Tuberculosis[1]

BY ISADORE KAUFMAN, M.D.

Of the many phases of the antituberculosis crusade, none to my mind is of more importance than the proper diagnosing of the disease. In order to obtain apparent arrest of pulmonary tuberculosis in any considerable number of cases, it is imperative to begin treatment in the early stage and, unless this diagnosis has been made, the patient will often drift until the disease becomes moderately advanced or even far advanced. It is obvious that

[1] From the Henry Phipps Institute of the University of Pennsylvania.

acute miliary tuberculosis is an exception to this rule when it is not superimposed upon a previously existent localized process.

The University of Pennsylvania endeavors to give its students the opportunity to obtain this instruction at its tuberculosis department, the Henry Phipps Institute. Students of the third-year class are divided into four sections, each of which comes to the institute one day a week for eight periods. One and a half hours is the time allotted for each instruction, or a total of twelve hours, in which to give such a course. Previously, my section has consisted of 15 to 17 students, but additional instructors this year have reduced the number of students to 10 or 12. However, I am following the same method of instructing which has apparently yielded results during the past five years.

Using three adjoining rooms the section is divided into small groups, and I am able to keep in close touch with the work of each student, who is soon made to realize that close attention to all details is essential for him to obtain benefit from the course. One or more patients, selected from the out-patient department, are studied in each room. The material for each demonstration is gathered with a particular purpose in mind, and each one represents a thoroughly studied patient by the instructor.

The method employed may be described as a building up of a diagnosis step by step and later the study of cases as a whole. Each instruction will be described in detail and you will gather my meaning of the method. Some phases of diagnosis are more easily grasped than others, so that I begin with changes in the spoken and whispered voice while percussion is considered last. During the first six instructions I am constantly with the students, and every step of their work is closely supervised. A few graduate physicians study in the clinics on other days and I find the same system of great benefit, although they can advance more rapidly and the lecturing can be omitted by referring them to text-books.

The first instruction concerns the demonstrating of changes in spoken and whispered voice sounds in pulmonary tuberculosis. This is preceded by instruction in the fundamentals of conducting

an examination with especial reference to proper position of the patient, removal of all clothing to the waist and observance of rigid silence in the room. The spoken voice is compared from side to side, beginning at the bases and proceeding to the apices, and then proceeding from base to apex on either side. I show the students how to handle the stethoscope and the method of proceeding in the examination, noting with a skin pencil the areas in which changes from the normal are present. As a rule, advanced cases are used, especially those showing but slight changes on one side, which is pointed out as the more important to be studied. After each patient has been demonstrated to the group in that particular room, the subdivisions in each room change about until each student has had the opportunity of eliciting the voice sounds in each of the 3 patients.

In studying whispered voice sounds the student is shown how quietly the patient must whisper and the differences of intensity of transmission in case of infiltration, consolidation or cavity, whispering pectoriloquy being designated for dense consolidation or cavity. This sign is likewise elicited by beginning at base and advancing to apices, but the comparison from side to side is unnecessary. The histories of the 3 patients are now given to the students in a general group, and it is pointed out to them that the proper taking of a history is most important, especially since the diagnosis of early tuberculosis must frequently be made from history rather than from physical examination, *x*-ray or other laboratory studies. This is followed by a brief talk on the surface anatomy of the lung, with especial emphasis upon the apices, limits of bases and the fissures.

The second meeting of the section is principally occupied with a study of the rales in pulmonary tuberculosis, and I teach that there are two great subdivisions of these adventitious sounds, crackling and whistling. The former are heard usually when produced in the alveoli or pleura, while the latter are produced in the bronchi. They are further subdivided into fine, medium and coarse, the number of rales being indicated by the terms occasional, few, moderate or many. It is my endeavor to point out rales in various

stages of tuberculosis, showing the coarser rales in the more advanced cases, but of course not omitting the instruction that rales may be absent in a very obvious case of the disease.

Especial emphasis is placed upon rales which are elicited only with cough, for this is so often the happening when studying cases of scattered infiltration, especially in the apices of the upper lobes. The cough must be properly used to be of value.

Cases with whistling rales are exhibited along with those of the crackling variety, so that the students may know the characteristics of each. It is pointed out to them that whistling rales are usually bilateral and general, occur in both inspiration and expiration and may disappear after cough. Crackling rales, unless the disease is in the pleura or massive in the lungs, occur often only with cough and are heard best with the cough or the very beginning of inspiration, are localized usually and rarely disappear with cough. When ulcerative disease is present the rales usually shade off in intensity and number from the apex toward the lower limit of the lesion, although any type of rale may be present in any given case. Also, it is pointed out that tuberculosis is not infrequently accompanied by a bronchitis and both general types of rales may be present at the same time.

As in the previous instruction, each student auscultates for rales each of 4 or 5 patients, who have been carefully selected for the demonstration, and emphasis is placed upon the regions where occasional fine, crackling rales are elicited. Each student is given the opportunity to test one advanced case of tuberculosis for changes in spoken and whispered voice. Then the section is assembled, when the histories of the cases are studied in detail and the complete diagnosis is given.

Following this a brief talk is given upon the abrupt onset of pulmonary tuberculosis, with especial emphasis upon hemoptysis and pleurisy, with or without effusion, as being presumptive evidence of tuberculosis, unless some definite cause for the conditions has been discovered. Also, the students are instructed as to the frequency of fistula-in-ano and ischiorectal abscess without obvious cause being of tuberculous origin, and how often a pulmonary lesion coexists or may later develop in that patient.

At the third instruction, especial study is directed to variations of breathing in incipient and advanced cases, with emphasis upon the former group. The students are shown cases showing variations from the normal, chiefly slight prolongation and blowing of expiration, while other cases showing granular breathing or even slight suppression of breathing are exhibited. I try to avoid the term bronchovesicular, but impress upon the students the necessity of recognizing slight changes in either inspiration or expiration, or both.

Alongside of these cases are shown patients demonstrating advanced lesions where blowing to both inspiration and expiration with a definite pause between the two is present. I make no attempt to distinguish between bronchial, amphoric and cavernous breathing, but am content to point out qualities of breathing which indicate advanced pathology. Three cases of advanced tuberculosis, with only a slight amount of disease on one side, are selected for this demonstration and the entire section auscultates each patient. Also opportunity is given to briefly review the instruction of the preceding periods.

Then the histories of all cases are given to the section, followed by the anatomical diagnosis. After this a talk is given upon the insidious onset of tuberculosis, with the modes of abrupt onset again reviewed.

The principal subject of study at the next session of the section is a consideration of inspection and palpation. One who sees any number of truly incipient cases of pulmonary tuberculosis knows that frequently all physical signs are negative, or that only a few fine crackling rales after cough are present, or possibly granular breathing is the only sign elicited. Therefore, generally speaking, inspection is not usually of value in the diagnosis of early lesions.

In cases of long standing, even though of slight extent, localized retraction, diminution of motion or lagging, can be pointed out. There is no question that most every moderately advanced case of pulmonary tuberculosis exhibits inspection changes which are very definite to any student. Nothing can be more easily demon-

strated than the changes on inspection which are revealed in cases of moderate pleural effusion.

Tactile fremitus, while the least valuable of all physical signs, is pointed out to the students in all 3 cases. The opportunity is given to review all the physical signs taught in previous instructions, after which the histories of the cases studied are reviewed, and especially are the modes of onset discussed. Following this a brief and to the point talk is given upon the symptoms of tuberculosis and the diagnosis without physical signs is emphasized.

At the fifth session of the section the difficult subject of percussion of the lungs takes up the major portion of the hour and a half of study. It is generally recognized that percussion of the lungs is the most difficult of all physical signs of the chest to properly estimate. The students are taught to pay especial attention to the length of notes as well as resistance to the percussed finger. At the outset they are shown the comparison of percussion notes over base of lung, over thigh and over trachea. Having this as a foundation, the students begin with percussion of bases anteriorly, noting the character of the findings and the similarity of the two sides. Assuming that the cases selected do not show basal changes, percussion is practised from side to side and approaching the apices, changes in length of note and resistance to finger being pointed out. Then percussion from base to apex on each side is performed, and changes found are thus compared with the normal. The slight unilateral changes are often detected in this manner. Each student then reviews voice changes, rales, breathing, inspection and palpation of their original cases, after which the abnormal percussion areas of the other 2 cases are pointed out to each student. The students, as a group, are then given the histories and diagnoses of the cases studied, and this is followed by a brief talk on the pathology of tuberculosis.

For the sixth session of the section, 3 cases showing slight unilateral disease are selected, and I go through the examination step by step, recording on a chart the findings just as they are obtained. After noting spoken voice changes in the case in the first room the students check up this finding while I am making

similar examinations in the other two rooms. Each feature of physical diagnosis of the lungs is similarly recorded and pointed out until the examination is completed. Near the end of this part of the instruction each subsection is given the history, laboratory findings and diagnosis of the case under study. Following the complete recording of physical findings by each subsection the entire group is given a talk on prevention and prophylaxis of tuberculosis.

At the seventh meeting of the section the students are divided into groups of three each and cases of a similar type to those used in the preceding instruction are examined by the students without assistance from me. Each subsection is given the complete history and laboratory findings of their individual cases. The students must make out a chart, recording each phase of the examination at its completion, but not after the entire physical findings have been tested. When the charts are completed I compare the findings of the students with my own data and point out any discrepancies. This takes up the entire time of the section.

At the eighth and final session of the section the students make out their charts on a similar type of cases, the only assistance given being the histories and laboratory findings of their individual cases. As each subsection is finished its examination, any discrepancies in my own findings and its are pointed out. At the completion of the chartings the entire section is given a brief talk on treatment, which emphasizes the importance of proper rest and feeding in pulmonary tuberculosis. It is my endeavor to point out to the students which types of cases can be successfully handled by general practitioners at home and which types should be referred to specialists or to sanatoria.

Throughout the course the students are taught the necessity of proper examination of the sputum, and they are urged not to neglect this procedure, irrespective of the diagnosis made. The importance of *x*-ray study is stressed, but the students are taught that it is secondary in importance to history and physical examination in the diagnosis of early or slight tuberculosis. The subjects of tuberculin and serological tests are briefly mentioned to acquaint the students with their place in diagnosis.

Conclusions. 1. To teach the diagnosis of pulmonary tuberculosis to a group of 12 to 17 students in a very limited time the above method in my hands has proven superior to any method of teaching of the subject.

2. The building up of a diagnosis enables the student to appreciate the importance of each step of the physical examination of the lungs.

3. The recording of each part of the examination as completed, emphasizes the importance of careful, painstaking work.

4. The students have been almost unanimous in their interest and, in after years, have expressed the thought that the course has been of great benefit.

DISCUSSION

Dr. Ward Brinton: It is a great pleasure to have heard Dr. Kaufman's very admirable paper and he has shown us the necessity of thoroughness and care in our methods with students. We are as physicians all of us teachers, of course, and since we are all teachers it is a good thing for us to know how best to teach. Furthermore, all of us are students, for no doctor can ever claim to know everything, and so it is a good, indeed a necessary thing, to know how to be taught. In regard to the special subject covered by Dr. Kaufman's remarks, there is a fault which I think many of us teachers in physical diagnosis make, that is, we try to cover too much ground too rapidly and to too many students. If there is any mistake in Dr. Kaufman's method I should say it is that, instead of having sections of eight or ten or twelve, we who are trying to teach physical diagnosis should plead with the colleges to give us smaller sections. I personally hate to instruct more than four men. I like to have that section over and over again because I am convinced that in teaching chest sounds as in teaching music it requires practise and still more practise. Ole Bull, the great musician, said, if I quote correctly, that if he did not practise any one day two fingers knew it, if he did not practise for two days three fingers knew it and if he did not practise for three days his whole hand knew it. That applies to us and our students. We have to give them a short course in a short space of time and it requires concentration and practise—as an example it is difficult to drive into the average student a thorough understanding between quality and pitch, and I have been ashamed of myself and my teaching ability over and over again when as I have thought I had covered the subject fairly thoroughly and then toward

the end of the class have a student ask, "Now, Dr. Brinton, what do you mean between quality and pitch?" Indeed, I have felt like throwing up my hands and saying, "Mon Dieu!" There is one element which is neglected too often I think, and that is the practical association in the student's mind between the pathological and clinical. We must remember that physical diagnosis is made up first of all by eliciting signs properly. In other words, if there is impairment or dulness the student should elicit the sign and say impairment or dulness. Then the sign must be interpreted correctly, for if there is an improper interpretation diagnosis will be in error. Again, I plead with the colleges that they should pay greater attention to the postmortem or necropsy in relationship to the clinician. Seldom do we see him standing alongside the necropsy table with his class, and as the pathologist lifts the lungs, let us say, from the thorax and explains the pathological condition, that he then, with the students around him, says in some such words, "Yes, gentlemen, the pathologist has shown you that here is the cavity, the physical examination says there was cavernous breathing and whispering pectoriloquy, and now we see the actual cavity in the lung." I think the perfect clinician is the one who has the power, through long practice, and by observing the findings on the postmortem table, to visualize the lung. When I hear a rale I say, "What is a rale? It is moisture. What kind of moisture is causing this particular sound I am hearing in this particular chest at this time?" One of the things I request of my students is that they take up the sputum cup by the bedside and look into it, and after doing so I say, "You see that material in the cup, note its character and its consistency. Now take your stethoscope and listen to the chest and picture that the material in the cup is causing the rales you hear." So I try to make them associate the condition with the sign and visualize. Again, perhaps, when at the postmortem there is noted roughened pleura or fine adhesions—I say here we have friction sound. Practically in no case, I explain to them, are you able to pass your hand around the lung within the thorax without getting a tear or finding an adhesion of some sort. "What can that show? That can cause friction." In other words, I would like to see more attention paid to the practical side of the pathologist's and clinician's work around the postmortem table in its relationship to physical signs and diagnosis. What Dr. Kaufman has presented is to the point that we should and can teach our students care in diagnosis. The methods he has pursued we have all of us used; they are not new, they are your own; but it is the care in determining these signs, the order in which they should be taken up, and the thoroughness of their study which he emphasizes. Do not think you can teach a student rales, fremitus or resonance in an hour. I know I cannot, and I know Dr. Kaufman must feel at the end of his course that his students are just on the borderland of learning.

Dr. Elmer H. Funk: Some years ago I ran across the maxim that a course in medicine was good or bad in direct proportion as the course in tuberculosis diagnosis, and I believe that is true. I believe Dr. Kaufman has shown us how a good course in medicine can have a splendid course in tuberculosis. It is unfortunate the hour is late, because he brought up many things worthy of discussion. I know him very well, so if I direct somewhat personal remarks to him you will know they are based on friendship. I believe the way to percuss the chest is from above down and not below upward. He says these men are to be taught personally. I was taught tuberculosis by perhaps seeing one case in clinic and hearing a whole hour's lecture about it. Nowadays the men are getting instruction by men who say "here is a whispering sound," or we speak of "whispering pectoriloquy." It seems to me this is the great value of present-day instruction (Dr. Kaufman and I are in total agreement). In order to teach tuberculosis the medical school must have a tuberculosis unit and should have a ward of graded patients, and this has worked out satisfactorily in our experience. A group of men are assigned to a patient; they spend ten weeks in department, four hours a week, forty weeks. The wards being for advanced cases have every now and then a death and postmortem, so we can count on an average of two postmortem a month. These students assigned in this ward are bound by the period to have covered eight or ten patients, and are bound in course of that to come from ward to postmortem room. In that way you drive home a lesson taught from ward to postmortem. We find that this postmortem experience emphasizes many lessons we are unable to drive home at the bedside. Take these students to patients with advanced disease first. It is folly to teach regarding early signs of tuberculosis to the man. Teach him obvious things, large cavities, and gradually train him down to the finer things, and above all things try to stress to the student that the diagnosis of tuberculosis is not made wholly on examination. A curious inheritance consists of men who graduated years before who come back saying, "We would like to stay with you a week or so and perfect in diagnosis of tuberculosis." Then we vision trying to perfect them in physical methods. They come back thinking we may have some other uncanny method of eliciting rale. They have history, symptomatology, physical examination, sputum and perhaps x-ray, and to teach the student exclusively the physical diagnosis is to teach it properly. They do not have the time to make the sputum studies. I think Dr. Kaufman's paper represents the modern concept of those who are interested in tuberculosis in its proper presentation.

Dr. Daniel M. Hoyt: I do not think I can add anything to the discussion of Dr. Kaufman's most interesting paper, and I think he has

shown us what can be put into an extremely brief period. What has been emphasized by those who have preceded me is that the time is all too short to cover the subject matter which is necessary in a course in tuberculosis. I have been lately in New Haven taking up postgraduate work there, and have been very much interested in perhaps a slightly new method of study. What seems to me most difficult to get students interested in is the normal chest. They always want to study the pathological chest. I think more time in the preparation of the student for tuberculosis work should be given to the normal chest and the normal physical signs. Now, in New Haven they handled things in a little different way. For instance, they took 5 cases and emphasized just one thing, auscultation. You found a case in which you had typical bronchial breathing, we will say, and you found another case in which you had vesicular breathing. Still another case in which you had bronchovesicular breathing, etc. Now, taking a series of cases like that and putting a mark on the chest and taking a group of students and saying, "Now, go and listen to that breathing, only listen to that one thing, breathing, and write down what you think regarding that breathing as to its intensity, as to its pitch, as to its duration and as to its quality, taking into account both the inspiratory and the expiratory sound. Based on these things, come back and tell us what sort of breathing you think it is." Now, you give these students a certain amount of time to study that breathing, you then bring them back and they tell the type of breathing that it is and why they think it is so. Then take these same students to the x-ray room and show them the stereoscopic plates. Let the x-ray instructor show the student the exact area that he has been listening to and instruct him in what is shown in the x-ray plate. Now, to me this sort of thing was extremely valuable, if you have the time to do it, because the thing that is required in this work is infinite attention to extreme detail, and by taking just one thing and emphasizing it the student acquires a standard. I do want to thank Dr. Kaufman for this paper, outlining the concentrated way in which the course is handled in the short time allotted.

Dr. E. J. G. Beardsley: Dr. Kaufman's paper has given us an excellent idea of how well the subject, tuberculosis, is taught in the school in which he is a highly valued teacher. The method, as outlined, is certainly admirable and seems as nearly perfect as can be a method that is, of necessity, taught in so few hours and with so few skilled instructors available.

In Philadelphia we are less fortunate in obtaining large sums of money for the endowment of our medical schools than are the schools of many medical centers. We have, however, reason for just pride in the well-deserved reputations sustained by our clinical teachers. Could we obtain

a larger supply of teachers like Dr. Kaufman the reputation of Philadelphia as a medical center will continue to be what we all wish it to be.

That a good medical school under modern conditions must possess self-sacrificing teachers is evident. Dr. Kaufman, and every other efficient clinical teacher of our city, is the poorer, financially, the more efficiently and self-sacrificingly he devotes his time to teaching. When one remembers, however, the tremendously valuable services that a clinical teacher renders his school, the students that come under his influence and the profession of which he is a member, one cannot but envy him the joy and satisfaction that come only to the true teacher. One of the real problems of all medical schools is connected with the obtainment of a group of younger clinical teachers. These teachers cannot be secured, trained or developed in a short period of time. Where are the schools to obtain the younger group of teachers to replace the senior teachers in the future? Many of the younger practitioners feel that they cannot afford the time to train and to teach as their predecessors have done. To teach is a luxury and necessitates unselfish devotion to an ideal of service.

Every medical teacher devoutly wishes that medical schools could obtain large sums of money to be devoted to erection of new buildings, proper equipment and maintenance, and, in addition, would like to see proper recognition of the great value of efficient teaching methods of the clinical branches. No medical school possesses the financial ability to properly recompense the efficient clinical teachers, but one cannot but visualize the good that would result if a medical school could, at least partially, recompense the younger teachers for the time they spend in preparing medical students for professional lives.

Dr. Kaufman's paper has given all of us food for thought. While Philadelphia possesses teachers of his ability its reputation as a medical center will not suffer.

Dr. Kaufman (in closing): I am indeed gratified, if not overwhelmed, by the complimentary remarks that have been made in reference to the humble efforts that I have given to this work in the last twelve years. For the first five years of my teaching I taught in the usual way by giving two or three students a patient, allowing them a sufficient length of time to go over the patient and then go over the patient with them. It did not take me very long to learn the student obtained practically nothing from such a course. As to the size of the sections, I agree heartily with Dr. Brinton, the smaller the section the better the work; but if you have ten or twelve students to deal with what are you going to do? You have got to give them instruction the best way you possibly can. Back of all this we should remember that when the student comes to the third year he is

supposed to have had a course in physical diagnosis. That course in physical diagnosis supposedly has emphasized the normal chest. At the University of Pennsylvania, the same as all the larger schools, a number of students enter the college at the beginning of the third year, coming from other schools. We find that their education in physical diagnosis has been very meagre. They know very little of the method of diagnosis. It does not seem just the right thing that a man teaching tuberculosis to a group of third-year students, who are supposed to have had a course in physical diagnosis, should have to go into the details of physical diagnosis, and yet it is true that we must do it. At the University of Pennsylvania this year the faculty has decided to give more work in physical diagnosis. Last year and again this year, owing to the illness of Dr. Landis, I have been asked to give the lectures in physical diagnosis, and we are stressing during the first half of the year normal physical diagnosis. It makes no difference what part of the body we are dealing with the instructors have been directed to teach the normal the first half of the year and then the second half of the year a program will be mapped out whereby they can teach abnormal physical diagnosis in various regions of the body. I think this is a decided step forward. In regard to Dr. Funk's statement of conducting his physical diagnosis from apex to base, I think the majority of teachers are agreed that when you advance from normal to pathological it is much easier to point out pathology to the student than it is to go from the other direction, and when dealing with limited lesions, particularly is this so. I have frequently seen bilateral disease, moderate disease on one side and very slight disease on the other and comparison from apex to base, the side on which lesser amount of disease is present has been entirely overlooked. Therefore, I am still of the opinion if we go from base to apex we will obtain better results. The point brought up by Dr. Hoyt is not quite *apropos*. At the school at New Haven they were dealing with graduate physicians, some of many years' experience. However, I am sure some of them needed instruction in fundamentals, but when I am dealing with a group of students, some that have just passed their second year in medicine, I realize the problem is a little bit different though very often it is one and the same thing to deal with. There can be no question that the method that I have followed in this teaching does not make a man a skilled diagnostician. All I am hoping to do is to give the student a working foundation, and the results, as they are manifested by criticisms that come to me in after years, that this course has put them on the right track and that they have been able to make diagnosis because of this instruction, certainly encourages me to continue this method, at least for the present.

Dr. Herman B. Allyn: I am tempted to speak of the teaching of physical diagnosis of many years ago. At that time we spent a good part of the first year in teaching the normal chest conditions. I gave up teaching some years ago, and there has been time enough in the interval for a great many changes to have occurred; but I was not aware that the custom of carefully teaching students normal conditions first, before the pathological were presented, had ever been abandoned.

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Certain Considerations in the Diagnosis of Oral Foci in Infection

BY ROBERT H. IVY, M.D., D.D.S.

That an etiologic connection exists in many cases between chronic foci of infection about the roots of the teeth and disease elsewhere in the body is at the present time generally accepted. Laboratory and clinical studies which support this view form an immense mass of literature, with which I shall not occupy your time. When this connection first began to be intensively studied, opinion was divided into two camps—the skeptical or ultraconservative, composed of those who did not believe in it at all, and the ultraradical, consisting of those who attributed practically all otherwise unaccounted-for human ills to mouth infection, and advocated the removal of all pulpless teeth. At the present time the ultraconservative view is gradually disappearing and there now remain the radical advocates of removal of all pulpless teeth and those who take a sane, middle ground, weighing the evidence based upon careful examination and diagnosis in individual cases.

What are the types of chronic mouth infection that are sometimes responsible for metastatic involvement of other regions of the body, and in what way is this systemic action brought about?

In a general way it may be said that there are two types of lesions about the teeth which may be regarded as sometimes having

an etiologic bearing on certain forms of disease of other parts of the body. These are:

1. Lesions involving the periapical region of the tooth, dependent on infection following death of the pulp.

2. Lesions involving primarily the investing tissues of the teeth —the periodontal membrane and alveolar process—not dependent on death of the dental pulp, but in which the infection starts at the gum margin—so-called pyorrhea alveolaris.

A study of the pathology bears out the clinical evidence that lesions of the periapical type, draining directly into the lymph and blood streams without external outlets, are much more important sources of systemic infection than the pyorrheal lesions with pus discharging into the mouth. It is probable that few of the bacteria that are swallowed from this pyorrhea pus actually enter the system, the majority being destroyed in the stomach. What I shall have to say, therefore, will apply particularly to the periapical lesions, although in a consideration of the mouth as a source of infection the presence of pyorrhea should by no means be ignored.

Many clinicians are satisfied with classifying all of these periapical conditions under the general term "chronic abscess," and there is a widespread impression that actual pus must be present in the bone cavities about the ends of the tooth roots. As a matter of fact, these lesions exist in the suppurative stage in only a small percentage of cases, so that the term abscess is generally a misnomer. In the vast majority of periapical lesions the disease results from infection following death of the dental pulp, the causative bacteria passing up the root canal and gaining access to the periapical tissues through the apical foramen of the tooth. The lesions are practically never seen in connection with teeth whose pulps are vital. The infection may follow dental caries and inflammation and death of the pulp—death of the pulp from traumatism or deliberate devitalization by the dentist. Failure on the part of the dentist to observe strict asepsis in performing root-canal operations has in the past been a frequent means of introducing streptococci from the mouth surface into the periapical

tissues. This source is gradually being eliminated by improved technic. The original infection in practically all cases is streptococcic, the inflammatory reaction giving rise to little apparent disturbance, but is slowly progressive and proliferative in nature. There is, first, a thickening of the apical peridental membrane, which undergoes a round-cell infiltration. This thickening gradually increases at the expense of the bone of the alveolar process, and as proliferation of round cells occurs the bone becomes rarefied and finally destroyed (chronic rarefying osteitis), the result being a space filled with a mass of chronic inflammatory granulation tissue, to which for want of a better term the name granuloma has been given.

Microscopic examination of the granuloma shows that it consists of small round cells, polymorphonuclear and endothelial leukocytes, fibroblasts, and running through it in all directions are numerous capillary bloodvessels. From this tissue streptococci may be obtained both by direct smear and by culture. Through the capillaries and lymphatic spaces of the granuloma the bacteria may easily gain access to the general circulation. While the outer layers of the granuloma may be denser and more fibrous than the inner portion, tending to restrict local spread of infection, there is no limiting membrane in the sense of preventing its contents from entering the general circulation and being deposited in distant structures of the body.

Occasionally the granulation tissue breaks down and becomes replaced by fluid pus, which fills the bone cavity. In the suppurative cases a secondary staphylococcic infection has usually been superadded to the original streptococcus. These areas of bone rarefaction and destruction vary greatly in size, though the size of the area does not necessarily indicate the virulence of the infection present.

Frequently, cysts develop from these granulomatous areas, the origin of the epithelial lining being epithelial cell rests in the normal periodontal membrane, which are the remains of the original enamel organ. Stimulation of these dormant epithelial cells is brought about by the chronic inflammatory process. These dental

root cysts vary considerably in size, from that of a small pea to a hen's egg. The cyst fluid is usually clear and may be sterile, but secondary infection frequently converts it into pus. By expansion a cyst involving at first only one tooth may destroy pulp vitality of several adjacent teeth, converting them into secondary foci of infection. A cyst in the upper jaw may also encroach upon or open into the maxillary sinus or nasal fossa. Cyst formation can frequently be differentiated from granuloma by means of the *x*-ray.

These chronic periapical conditions may exist in the entire absence of subjective symptoms or physical signs. In order that serious errors may be avoided, cases of disease of other parts of the body in which dental infection may possibly be an etiologic factor demand careful examination of the teeth and jaws by all the clinical and laboratory means at our disposal and the search for and elimination of possible causes elsewhere. Loss of the teeth as masticating organs may possibly be a greater evil in some cases than the retention of a suspected but not proved focus of infection. Even though dental pathology be demonstrated, the injudicious removal of teeth in a case of severe illness may be the determining factor in a fatal outcome. These considerations render necessary a close coöperation between the internist and the mouth specialist. Many physicians in the past, without troubling about a clinical examination of the mouth, have had the teeth *x*-rayed by a general roentgenologist, who has, likewise without clinical examination, designated certain teeth as the seats of "abscesses," as shown by certain shadows marked by arrows on the film, and the physician has ordered these teeth to be removed. As a consequence, many people have been deprived of useful, vital, harmless dental organs without the least benefit to the general health. The diagnosis of peridental disease cannot always be made on *x*-ray examination alone, and the roentgenologist who has no knowledge of dental pathology or clinical examination of the teeth has no right to make a clinical diagnosis from his plates or films alone, and especially to furnish, as is sometimes done, unsolicited recommendations as to treatment.

It is unnecessary to more than mention the ludicrous mistakes that have sometimes been made, owing to ignorance of the normal anatomy of the jaw bones. Dark shadows in the films cast by the mental foramen, the maxillary sinus, the nasal fossa and other normal spaces have been mistaken for lesions due to dental infection. In marked cases of bone rarefaction about tooth root ends the *x*-ray alone is, of course, frequently sufficient for diagnosis; but sometimes the normal peridental shadow line of a vital tooth is slightly thicker than usual and can only be differentiated from the abnormal shadow of disease by careful clinical tests for pulp vitality. On the other hand, a tooth may contain a dead infected pulp which may be throwing streptococci into the circulation, and not be detected by the *x*-ray because there is no periapical bone absorption. For these reasons any examination for the discovery or elimination of dental infection as a source of systemic disease should consist, first, of a clinical examination of the mouth, with tests for vitality of pulps of doubtful teeth, followed, if necessary, by an *x*-ray examination of the teeth and jaw bones. If the clinical examination shows the mouth to contain a full quota of teeth with vital pulps and no pyorrhea, an *x*-ray examination will usually be superfluous in searching for infection. Teeth bearing crowns or acting as supports for bridges should always be under suspicion, but it should be noted that crowns and bridges are frequently attached to teeth with vital pulps, and therefore, *per se*, are not necessarily to be condemned. In cases where teeth have been lost the alveolar process may contain remnants of infected roots or foci of bone infection giving no clinical evidence of their presence. The *x*-ray is of great help in the detection of such conditions.

Where the presence of dental infective foci has been demonstrated, can we decide in that particular case whether these foci are doing any harm to the individual as a whole or are responsible for lesions existing elsewhere in the body?

Many attempts have been made to determine in individual cases whether or not periapical dental disease is associated with systemic reaction. In 1913 Hastings[1] performed complement-

fixation tests in arthritic patients having dental infective foci, using a polyvalent nonhemolytic streptococcic antigen, and reported somewhat encouraging results. Logan[2] found a leukocytosis in 47 of 52 cases of periapical infection. Probably the most favorable report from this standpoint is that of Daland,[3] who found in a large percentage of cases of chronic nonhemolytic streptococcic infection, from various sources, a lymphocytosis of small lymphocytes associated with leukopenia. Goadby[4] thinks that there is constantly an increase in the mononuclear cells of the blood in systemic disease connected with pyorrhea. Toren[5] has attempted to demonstrate the presence of large lymphocytes with a peculiar staining reaction in cases of oral infection, and claims that these lymphocytes are found only in such cases. He makes no effort to explain why these peculiar cells should result from streptococcus infection about the teeth and from no other source, and, unless confirmatory evidence is forthcoming, his statements should be accepted with great reserve. Haden,[6] after a study of the blood in 200 patients, concludes that the complement-fixation method has furnished little result in the diagnosis of these cases. Regarding the leukocyte count, he found that patients with periapical disease showed a slightly higher total white count than those having no periapical infection. The difference is somewhat more marked in those patients suffering from systemic disease of focal origin. He finds that the increase is for the most part in the polymorphonuclears and not typically in the lymphocytes. The study has demonstrated to him that the differential leukocyte count is of no definite value in the diagnosis of absorption from areas of chronic infection.

In view of these rather conflicting reports, it may be said that, so far, we can depend very little on blood examintion to determine in the individual case how much a focus of infection in the mouth has to do with a lesion elsewhere in the body. We can demonstrate the presence of these mouth foci, and we know by experimental and clinical evidence that they frequently cause serious metastases. These facts are sufficient warrant for eliminating them whenever they exist, whether the individual is otherwise healthy or not.

What shall be done in these cases when periapical lesions have been demonstrated?

The object of all treatment is the elimination of infection, whether it be by conservative treatment of the tooth, extraction or extraction and curettement of the surrounding area of bone disease. In the lesser grades of periapical involvement it may be conceded that infection can be cleared up with reasonable certainty by modern methods of root-canal medication. We have seen, for example, complete bone regeneration in large areas of periapical destruction as the result of medication through the root canal with dichloramin-T. It may require a considerable period of time, however, to determine the result of conservative treatment, and this waiting period may be fraught with danger to the patient. It is sometimes possible to save an upper anterior tooth otherwise doomed to extraction by removal of the apical portion of the root and curettement of the surrounding diseased bone. This, however should only be attempted after sterilization and filling of the root canal. The question of conservative or radical treatment of teeth showing only moderate periapical involvement should first of all be decided by the general health of the patient. Our attitude toward suspected teeth in patients who have some systemic condition, in which mouth infection is possibly playing a part, should be more radical than that adopted in patients having no physical ailments. Conservative treatment of the tooth may often be attempted in a healthy individual where in an invalid a tooth so affected would be removed without hesitation. In cases of extensive periapical involvement, whether in an invalid or an otherwise healthy person, there is no question as to the advisability of radical surgical treatment.

A word or two about removal of these foci of infection in the mouth. Where certain teeth have been condemned as a menace to the health, the usual dictum of the physician is for the patient to go and have them extracted, in the belief that extraction will end the trouble. That extraction alone is not sufficient in many cases is borne out by the almost every-day experience of discovering persistent foci of alveolar disease in people for whom tooth extrac-

tions have been performed sometimes years before. When the x-ray reveals the presence of a well-developed granuloma or cyst, extraction of the tooth should be followed by curettement of the diseased tissue contained in the bone cavity surrounding the root apex. It is true that often tooth extraction alone will enable Nature to take care of the remaining periapical disease, but it is equally true that healing of a tooth socket may take place over a mass of granulation tissue or a cyst, giving rise to serious local or general trouble.

Local anesthesia with novocain-adrenalin is the method of choice in all operations for the relief of these conditions. It restricts the amount of hemorrhage, giving a clear view of the operative field and the pathology present. After extraction of the tooth and curettement of the bone cavity there is ample time for trimming off and smoothing the alveolar process and approximating the edges of the gum tissue so as to ensure a smooth surface on healing.

Wholesale extraction of many teeth at one sitting in invalids is to be condemned. The shock of the operation may be severe, or a large amount of infection may be set free into the general circulation and greatly aggravate the general condition. Where there are many mouth foci present, two or three teeth should be dealt with at one time, and four- or five-day intervals allowed to elapse between the operations.

If necessary, cultures may be obtained from these foci of mouth infection, with a view to preparation of autogenous vaccines. Many times cultures taken from these cases are valueless, being contaminated from the secretions of the mouth. It is a most difficult matter to remove a tooth from its socket without having the sides and apex of the root come in contact with extraneous contamination. The most certain way to obtain a growth of the organisms responsible for the infection is to flame the extracted root with alcohol, cut off the apex and take the culture from the root canal thus exposed. While cultures are not obtained in such a large percentage of cases by this method as from the outer surface of the root of the tooth, when they are positive we can be reasonably sure that they are not due to contamination.

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DISCUSSION

Dr. Joseph Head: Dr. Ivy's paper is sound and conservative. I, however, am not so skeptical about the possibility of extracting teeth aseptically so as to get an uncomtaminated vaccine from the tips. This is especially so if the tooth is standing alone and the cautery is used around the neck or even iodine applied around the tooth carefully and then dried. After these precautions have been taken, if the tooth is extracted with sterile forceps I feel that the tip of the root in 90 out of 100 cases will be free from mouth contamination. In fact, when this method has been used I have sometimes succeeded in getting sterile results, the tip being free from infection and showing no growth when placed in the Noguchi tube.

This brings us to another point, which has long been a question, whether a tooth with a filled root can be sterile and free from any bacterial foci that might react upon the system. There was a tooth the root canals of which had been filled and an *x*-ray showed no evidence of apical or gingival infection. Two years later an *x*-ray film showed the gum to have receded posteriorly, so that the bifurcation of the lingual and posterior buccal roots had become exposed and that the infection had reached to the tip of the lingual root and almost to the tip of the posterior buccal root. The anterior buccal root seemed perfectly sound and free from infection. The tooth was extracted aseptically and the three tips put into separate Noguchi tubes. The culture in which the anterior buccal tip had been placed was sterile, the other two tips showed the presence of hemolytic and non-hemolytic streptococci. This would seem to indicate that a pulpless root tip may be sterile.

There was another point that Dr. Ivy did not have time to bring out in his admirable paper, and that is the danger of leaving impacted teeth in the jaw. Impacted teeth seem in some mysterious way to develop toxins that permeate the system and cause serious derangement of the nerves, kidneys, heart and joints. Although I have extracted them and had them examined bacteriologically, I am not convinced that the bacterial infection can sufficiently account for the grave systemic disorders

that disappear when these teeth are extracted. It has occurred to me that the inhibition of the tooth development may have an analogous effect to the inhibition of the development of the embryonic connective tissue, for just as the toxins of the sarcoma pass through the system apparently due to the lack of development in the embryonic connective-tissue cell, in a similar way the unerupted tooth may develop a characteristic poison of its own.

It has been my clinical experience that the removal of impacted teeth after the age of thirty or forty years, when they are associated with serious systemic disorders, is also usually associated with marked improvement.

Dr. James R. Cameron, D.D.S.: Inasmuch as the hour is late, I shall just confine myself to one phase of the paper. I believe Dr. Ivy has presented to you a paper very sane, not going to any extreme, and the chief value of the paper, as I see it, is that it points out to you as medical men the great need for coöperation between medicine and dentistry. I believe the time is coming when a man taking up the study of dentistry will first study medicine and obtain the medical qualification, because today no matter how you look at dentistry it is the practice of medicine to a limited field. The problems are overlapping all of the time. There is one phase I would like elaborated a little more, although Dr. Ivy has touched on it, that is the interpretation of diseased conditions of the teeth from *x*-ray plates alone. Frequently not only physicians but dentists will refer cases to commercial *x*-ray laboratories, to men who are neither dentists nor physicians, and it is a joke to read their reports, and spells disaster for the patient.

That is most misleading when you examine a mouth and teeth you will find the *x*-ray is very much in the dark. The radiograph is a wonderful aid in diagnosis, but does not supplant a clinical examination.

Dr. Myer Solis-Cohen: I would like to ask Dr. Ivy a question. It has seemed to me that where a root has been properly treated, infection of the respiratory tract may sometimes cause secondary infection at the root of the tooth. I frequently have found the same organism in the root of a tooth and in the tonsils, and it has seemed to me that persons with tonsillar infection, or infection of the upper respiratory tract, are more liable than others to dental infection. I have wondered whether the dental infection could not be due to the tonsillar infection, and that instead of blaming the dentist for poor work it might not be the case that he has done good work and reinfection has occurred through the blood stream. I would mention a method of culture I employ. The dentist removes the tooth and an assistant holds it in the forceps while I take a sterile swab—cotton on an applicator—and make a smear from the apex of the tooth,

which I rub both on an ordinary culture medium and on the bottom of an empty sterile test-tube. I then draw 5 or 6 cc of fresh blood from the patient's arm and place it in the empty test-tube, overlaying the culture. The blood has bactericidal power against the organisms that are non-pathogenic to the patient, and in consequence destroys all contaminations by organisms not pathogenic to that patient. Those that grow up in the patient's blood are pathogenic to him. Five or six organisms may grow up in the ordinary culture tube and only one or two in the patient's own blood.

Dr. Ivy (in closing): I was interested in what both Dr. Cohen and Dr. Head said about culture methods in these cases. The ordinary method of making smears from the root surface after it has been extracted, without special precautions, should be discarded. It is quite possible that teeth may be secondarily infected from diseased tonsils. We know that teeth whose pulps have been devitalized by trauma may develop areas of apical disease in the absence of any break or decay of the surface of the tooth. Here infection must be carried to the root end through the blood or lymph streams. In such cases it is conceivable that in teeth with previously devitalized pulps the periapical region may become infected by metastasis from the tonsil.

DECEMBER 17, 1923

Quinidine Treatment of Extrasystolic Arrhythmias

BY JOHN H. MUSSER, JR., M.D.

(*Abstract*)

Much attention has been paid to the use of quinidine in the treatment of auricular fibrillation. In certain types of auricular fibrillation the drug proves to be of value. Little attention has been paid to the treatment of other types of cardiac irregularities with quinidine. In the literature there is only an occasional mention of its use in paroxysmal tachycardia, auricular flutter, extrasystoles and paroxysmal auricular fibrillation. In a series of cases the drug has been used with a great deal of benefit, particularly in the treatment of symptomatic extrasystoles. It is also of benefit in paroxysmal tachycardia.

DISCUSSION

Dr. Charles C. Wolferth: Dr. Musser's statement about the patient whose history he and I have recorded reminds me of a famous case of auricular flutter and heart-block which has been reported at least six different times—perhaps Dr. Musser's and my case will eventually be reported that often. It is a very instructive case, because it demonstrates the possibility of controlling paroxysms of auricular tachycardia by the use of quinidine.

The thing about this patient so favorable for study is that, when she is not under the effect of quinidine, she has short paroxysms every few minutes to a half-hour apart, as long as she is up and about. If she goes to bed the paroxysms disappear. Under my own observation her arrhythmia was completely abolished by quinidine seven times. In each instance the cardiac mechanism was maintained normal as long as desired. This is the only case of paroxysmal tachycardia I have had the opportunity of studying sufficiently to be able to say flatly that quinidine controlled paroxysms. There are cases reported, both of auricular and ventricular tachycardia, in which individual attacks are said to have been terminated by quinidine and their recurrence prevented by continued use of the drug.

Harvey and I, at the University Hospital, have had the opportunity of studying the effect of quinidine in a patient with numerous transient attacks of auricular flutter. These studies will be reported in detail later, but it is perhaps worth mentioning here that we obtained clear-cut evidence of an effect from quinidine of suppressing paroxysms. I have also observed what I have regarded as favorable results in paroxysmal auricular fibrillation, but, as in most of our cases of auricular tachycardia, the paroxysms did not recur frequently and constantly enough to admit of definite conclusions.

A point that may be of more than academic interest is the question as to the mode of action of quinidine in suppressing these forms of rapid heart action. Lewis has advanced a hypothesis for its action in suppressing continuous flutter and fibrillation which accounts for the results on the basis of changes in refractory periods in relation to changes of conductivity of auricular muscle rendering further circus movement impossible. Experimental and clinical data appear to confirm amply Lewis' view.

On the basis of evidence whose presentation here would be inappropriate, the hypothesis may be put forward that isolated auricular extrasystoles, paroxysmal auricular tachycardia, auricular flutter and auricular fibrillation all originate by the discharge of an excitation wave prematurely from an ectopic auricular focus, the subsequent course of events being

determined by the state of the functional properties of the auricular muscle being excited. Among these may be mentioned excitability, conductivity and refractory period.

Evidence has been presented tonight demonstrating the effect of quinidine in suppressing premature contractions. Others have reported similar results, so that we may regard the tendency as definitely established. Moreover, these results would appear to be in accord with the experimental demonstration that quinidine raises the threshold of stimulation necessary to evoke auricular contraction. Thus we may have an additional point of attack by quinidine in suppressing paroxysmal as compared with continuous arrhythmias, since the premature contractions initiating paroxysms may be abolished. This is what must have occurred in the case of paroxysmal auricular tachycardia, since all premature contractions were abolished by quinidine and no paroxysms had a chance to occur. In our case of transient paroxysms of flutter, the frequency of premature contractions was diminished, but they were not entirely suppressed. But whereas before the use of quinidin nearly all were followed by short runs of flutter after the use of the drug, isolate extrasystoles and extremely abortive attacks of flutter were the rule. Thus it would appear that quinidine was exerting an effect, on the one hand, of suppressing the initial disturbance, and on the other hand of making more difficult the maintenance of arrhythmia, once it had been established.

Dr. Musser has done us the service of directing our attention to a field of study and possibly therapeutic usefulness of quinidine, which, although not entirely neglected, has not had the attention it deserves. I wish to state my agreement with his discussion of the dangers of the drug in extrasystolic arrhythmia and paroxysmal disturbances as compared with those encountered in continuous auricular fibrillation.

Dr. James E. Talley: There is a copy of Briquet's exhaustive work on *Cinchona*, published in Paris in 1853, in the library of the College of Physicians here. This author showed that both quinine and quinidine slowed the pulse, but as he was dealing largely with patients who had a regular rhythm, the degree of slowing was not great, so aroused but little interest. I have had some luck with quinidine in patients with extrasystoles frequent enough to give discomfort. In others it has failed. Often this disturbing arrhythmia has been in those under stress, most frequently mental and sometimes physical as well. In these patients I suspect the rest was more potent than the quinidine. It is an occassional experience to convert auricular fibrillation into auricular flutter under the influence of quinidine and have the flutter persist. In a recent patient with the clinical signs of failure by congestion and a regular apex-rate too high for ordinary clinical flutter, the giving of quinidine was followed by a fall in rate and the electrocardiogram showed definite 2 to 1 flutter.

Unfortunately no electrocardiogram was procurable in the beginning, so absolute proof is lacking of the exact original mechanism, though it suggested simple paroxysmal tachycardia.

Dr. Ross V. Patterson: It seems to me there are several angles to the use of quinidine in cardiac arrhythmias. I have been much interested in Dr. Musser's presentation of the subject. I would emphasize the difficulty of determining the value of any remedy in an intermittent and irregular disorder such as premature contractions or paroxysmal tachycardia; conditions which, of course, have a spontaneous tendency to a termination, certainly require very close observation in a considerable number of cases to decide whether a given remedy has any constant effect or whether in many cases the termination of the attack might be due to a spontaneous tendency to return to normal. In the second place, it seems to me that the question of the real value of the drug in these cardiac patients should be considered. The real test as to the value of any remedy is whether the cardiac efficiency has improved under its administration or whether it has not. Quinidine must be a protoplasmic poison. It is owing to this effect that it has the value in malaria; it must have a specific depressant effect upon the heart, its increase of heart-block, the conversion of heart-block into a higher grade is an aid as to understanding its effect upon the conduction system. In conditions such as premature contractions their mere incidence certainly would not indicate the necessity for administration of any remedy. There may be a lot of extracardiac causes or intrinsic causes. If their occurrence does not produce distress to the patient it is doubtful whether any treatment is indicated, since the cardiac efficiency is little if any impaired by their occurrence. In these cases, where the extrasystole arises from the intensive cardiac damage, it would seem dangerous to administer a cardiac depressant in large doses. I believe that the whole question as to the value of quinidine in cardiac cases is still *sub judice* and will require considerably more observation to demonstrate whether it has a real value or whether it merely controls a symptom without actually improving cardiac condition. Indeed, in many cases it perhaps impairs the cardiac condition rather than improves it.

Dr. Musser (closing): I did not mean to advise the use of quinidine in every case of extrasystolic arrhythmia, because the great majority of patients suffering with this irregularity are not even cognizant of the fact that they have it; but I think that the drug is indicated merely in a certain number of cases. In regard to the dangers connected with it, I feel that this danger has been exaggerated. After all, quinidine is a salt of quinine, and we have used quinine for a great many years, and I do not think there is much danger, if the patient is carefully controlled and watched, in the treatment of extrasystolic arrhythmia with quinidine.

The Treatment of Goiter, based upon its Physiology and Pathology

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The normal thyroid secretes a substance which is delivered into the circulation, and which, according to Plummer, is essential to cell metabolism. When the gland has a derangement of function produced by stimulation, lack of an available chemical substance, or by a pathological process, it produces certain recognized diseases. In discussing a disease of the thyroid, a term descriptive of the clinical manifestations rather than the pathological changes should be used by the clinician, such as in the following classification:

1. Cretinism.
2. Myxedema.
3. Colloid goiter, congenital, adolescent or of pregnancy.
4. Exophthalmic goiter, acute or chronic.
5. Adenomatous goiter, with or without hyperthyroidism.
6. Thyroiditis.
7. Tuberculosis.
8. Carcinoma.
9. Sarcoma.

Of these diseases of the thyroid, the most frequently seen are colloid goiter, adenomatous goiter and exophthalmic goiter. These three forms will constitute the main subject of our discussion, but it will be necessary to review the physiology and pathology of the thyroid as the basis for the treatment.

Physiology. The primordial thyroid was an exocrine gland which delivered its secretion through the thyroglossal duct, and this primitive stage of the gland is found in the lower vertebrates, such as the larval stage of the lamprey eel. In these lower vertebrates the thyroid duct closes down to a small pore, and it is not known whether the duct remains patent during the adult stage

of the organism. In all the higher vertebrates the duct is open during the early life of the embryo, but soon closes by obliteration. When the thyroglossal duct was the channel through which the secretion from the gland was delivered, all the secretion passed from the cell into the acinus. With the obliteration of the duct and the conversion of the gland into an endocrine gland, it is obvious that the mode of delivery of the secretion for utilization by the organism would have to be reversed, in order that it could be delivered into the circulation from the base of the cell.

Biondi, in 1892, was the first to observe a substance in the cells of the thyroid which reacted to stains in the same manner as the colloid stored in the acinus. He showed that the colloid found in the acinus was a product of the secretory activity of the acinic cells, as he found globules of colloid in these cells. Bensley, in 1916, found, by a special staining method, the base of the acinic cells of the thyroid filled with a sky-blue staining material contained in vacuoles. He considers this substance to be the secretion of the acinic cells. From his studies he concludes: "We may assume, therefore, that the thyroid gland, as all physiological and clinical experience indicates, prepares and secretes into the vascular channels of the gland a secretion, and that this secretion is formed in the outer pole of the cell and excreted from it directly under normal conditions of functioning without passing by the indirect route through the follicular cavity." Though highly specialized as an endocrine gland in adult life, the primal secretory function of delivering the glandular secretion through the thyroglossal duct is made use of in storing colloid. This product of the acinic cells, which is stored in the acinus, resembles the secretion which is delivered into the circulation from the base of the acinic cells. The presence of colloid in the follicular cavity and its variability has caused much speculation as to its origin and utilization. Bensley's studies of the secreting thyroid prove that this stored colloid is secreted by the acinic cells and may be withdrawn from the follicular cavity. Whether this colloid substance in the acinus is the finished product or whether it is a substance

which can be quickly utilized by the cell in making the secretion is not known. Resorption of this material from the lumen of the acinus does take place under conditions where the normal direct secretory activity of the cells is insufficient to meet the physiological demand.

Impulses are transmitted to the glandular epithelium of the thyroid by the sympathetic nervous system. The nerves of the sympathetic system supplying the gland are from the cervical sympathetic ganglia and accompany the bloodvessels by forming a plexus in their outer wall. The branches accompanying the superior thyroid artery are from the external carotid plexus which is formed by branches from the superior cervical sympathetic ganglion. The branches accompanying the inferior thyroid artery are formed by branches from the inferior cervical ganglion.

Chemistry. The presence of an iodin compound in the thyroid was reported by Bauman in 1895. To this substance he gave the name of thyroiodin. Oswald, in 1899, reported his studies, which showed the iodin compound to be a constituent of the colloid, and to which he gave the name of thyroglobulin. Nothing further of note of the chemistry of the thyroid was learned until the chemical researches of Kendall began to appear in the literature in 1914. He succeeded in recovering from the thyroid in a pure crystalline state an indol compound containing 60 per cent of iodin, which he considers to be the active constituent of the secretion of the thyroid, and to which he has given the name of thyroxin. By his experiments he demonstrated that this iodin compound will increase the metabolic rate and produce hyperthyroidism. The severity of the symptoms produced will depend upon the size of the dose and the period over which it is given. If it is given in a dose sufficiently large, it will produce acute hyperthyroidism and death. Typical exophthalmic goiter cannot be reproduced experimentally by the administration of thyroxin. Clinically, thyroxin has been used most successfully and with striking results in the treatment of myxedema and cretinism.

Pathologic Physiology. Certain deviations of the level main-

tained under normal conditions may occur by an increase or a decrease in the stimulation of the thyroid or by the availability of iodin. Slight deviations either above or below normal will produce changes which are almost symptomless.

The demand upon the thyroid for its secretion to supply the needs of metabolism will be met only if the cells can manufacture a colloid of a certain viscosity. This viscosity will depend upon available iodin. If the iodin is not available, a thick colloid will be produced, which will be carried to the inner pole of the cell and delivered into the acinus. If the secretion is not supplied in response to the demand the stimulus will continue, resulting in a moderate hypertrophy of the thyroid cells and filling of the acini with colloid. Colloid goiter is a manifestation of this derangement.

In adenomatous goiter a hyperthyroidism slowly and progressively develops. This hyperthyroidism is probably produced by cellular activity of the cells within the adenoma or of those in the parenchyma surrounding the adenoma, and not by stimulation through the sympathetic nervous system or a lack of available iodin. The chemical composition of the secretion producing this hyperthyroidism is unknown.

In exophthalmic goiter the increased demand upon the acinic cells, by the stimulation to the thyroid, is so great that most of the stored colloid is withdrawn from the acinus in producing the increased amount of secretion to meet the demand. The cause of the derangement of function and the chemistry of the substance produced by the cellular activity in this disease is not known, but is likely that the secretion is so altered that it is toxic. The altered physiology present in exophthalmic goiter is not clearly understood, nor is the chemistry of the secretion of the thyroid in this disease know, and we may long await a solution of this problem.

Treatment. As the physiology and pathology of each of the diseases of the thyroid is different, we shall discuss the treatment of colloid, adenomatous and exophthalmic goiter separately.

Colloid Goiter. Colloid goiter is a bilateral, symmetrical enlargement of the thyroid occurring during fetal life, adolescence or

pregnancy, and is endemic in certain regions. This symmetrical hypertrophy of the thyroid produces a noticeable enlargement of the neck anteriorly. The gland is soft but easily palpable. Occasionally there may be a bruit and thrill of the thyroid arteries. In a case of colloid enlargement of the thyroid, with a thrill and bruit of the thyroid arteries and nervousness, the patient presents a clinical picture simulating hyperthyroidism. In differentiating a case of colloid goiter from a case of hyperthyroidism the determination of the basal metabolic rate is of the greatest importance.

Marine and Kimball have made a most valuable study of colloid goiter among the school children in a goitrous district. Approximately 50 per cent of the school children in the series examined over a period of three years showed a slight, moderate or marked enlargement of the thyroid. The enlargement is six times as frequent in girls as in boys. 2190 pupils were placed on a prophylactic treatment of 2 gm. of sodium iodid twice yearly, giving 0.2 gm. daily for ten days, during each period of treatment. Of these, only 5 showed enlargement of the thyroid.

2205 pupils, living under the same environment, were not given the prophylactic treatment. Of these, 495 showed enlargement of the thyroid.

1182 pupils having thyroid enlargement on first examination were treated as above stated. Of these, there has been a decrease of the thyroid in 773.

1048 pupils having thyroid enlargement were not treated. Of these there has been a decrease of the thyroid in only 145.

The studies of Marine and Bensley show, and the work of Kimball prove, that colloid goiter is due to a lack of iodin, and that if this substance is supplied the gland will return to normal unless the follicular cavities of the gland have been distended with more colloid than the cells are capable of withdrawing. Diffuse colloid adenomata may not respond to treatment with iodin.

Adenomatous Goiter. An adenoma of the thyroid cannot be diagnosed clinically until it is large enough to be palpated or causes a noticeable enlargement of the neck. Many small adenomata,

which are too small to be palpated during life, are found at autopsy. Observation by the patient or clinician is usually first made during puberty or early adult life when the adenomata ordinarly enlarge sufficiently to be noticed. Adenoma of the thyroid may be single or multiple, unilateral or bilateral, and may vary in size up to 15 cm. in diameter or larger. The adenomata may cause enlargement of the neck anteriorly or laterally, on one or both sides; or they may be substernal, interthoracic or retrotracheal. They usually enlarge sufficiently to be noticed during pregnancy or early adult life. Growth of an adenoma may be slow or rapid, and after attaining a certain size may remain unchanged for years. Enlargement, which may or may not subside thereafter, may be noticed during menstruation or pregnancy. A sudden enlargement of an adenoma may occur at any time. This sudden enlargement is usually due to hemorrhage in the adenoma. Rapid enlargement may be due to malignancy.

The onset of toxic symptoms due to adenomata of the thyroid is so insidious that they may be present for a number of years before being detected. After from five to twenty years' duration, symptoms of hyperthyroidism can usually be detected. There is a great variation in the severity of the hyperthyroidism and the amount of tissue damage. Nervousness, tremor, tachycardia, hypertension, myocarditis, dyspnea and edema are encountered in varying degrees of severity in the most toxic cases. Pressure symptoms from sudden or progressive enlargement, causing dysphagia, difficult breathing, choking and hoarseness, may develop at any time. The rate of basal metabolism is not high, as a rule, usually being about plus 30 per cent. Occasionally the acinic cells, in an adenoma with well-developed acini in an otherwise normal gland, may hypertrophy and by their cellular activity produce all the symptoms of exophthalmic goiter. In such a case the basal metabolic rate may be high. Exophthalmic goiter, with the characteristic changes in the parenchyma of the thyroid, may occur in an individual having an adenoma.

From a study of the altered physiology in adenomatous goiter we have learned that a progressing hyperthyroidism is slowly

brought about by cellular activity of the cells within the adenoma or the cells in the parenchyma surrounding the adenoma. From this it seems logical that by the removal of the adenoma the hyperthyroidism with its resulting tissue damage is prevented or the progress of the disease stopped.

Every case of adenoma of the thyroid ought to be considered potentially a surgical disease, but should be primarily a medical one. In young adults operation should be deferred unless the adenomata are causing pressure symptoms by retrotracheal, substernal or interthoracic position, or for cosmetic reasons. After the third decade of life the patient should be advised to have the adenoma removed surgically, so as to prevent tissue damage by a slowly progressing hyperthyroidism, which is characteristic of this variety of goiter, and which will eventually occur. Nervousness, tremor, tachycardia, hypertension, myocarditis, dyspnea and edema, which are encountered in the most toxic cases, should be treated medically until the patient improves sufficiently to be considered a good surgical risk. An *x*-ray study should be made to determine if the goiter is substernal or interthoracic. The curative treatment of adenoma of the thyroid is the surgical removal. No other treatment has proven effective.

Exophthalmic Goiter. Exophthalmic goiter usually occurs in middle life, but may occur in childhood. It is rather sudden in its onset or the development of symptoms is rapid. Marked exacerbation of symptoms producing a crisis is characteristic. The crisis may result in a fatal issue or may subside. Recurrence of a crisis may occur after several weeks, several months or a few years. Some cases run a chronic course and may remain undiagnosed until marked tissue damage has taken place. As a diagnostic aid a study of the basal metabolic rate is important. The rate of the basal metabolism is usually high—from plus 50 per cent to plus 100 per cent or higher.

From what we know of the derangement of function in exophthalmic goiter, we are of the opinion that the marked increase of function of the acinic cells of the thyroid in exophthalmic goiter is due to stimulation transmitted to the glandular epithelium by the sympathetic nervous system. It is reasonable to presuppose

that when the cause of exophthalmic goiter is known, or when the chemistry of the secretion resulting from the pathological physiology has been detemined, exophthalmic goiter will become a medical disease. Until we know the cause and have more knowledge of the chemical nature of the substance secreted by the gland in this disease, our efforts will be toward inhibiting the stimulation, reducing the blood supply and removal of part of the gland.

During a crisis the patient should be placed under medical treatment until the crisis subsides. A chronic case may be operated upon at any stage of the disease unless there has been serious tissue damage. A case with myocarditis, cardiac decompensation and edema should be treated medically until the patient has improved enough to withstand the shock of the operation.

Primary thyroidectomy may be done in a case having a mild degree of hyperthyroidism. The usual surgical procedure in a case with marked hyperthyroidism is the ligation of the left superior thyroid artery, followed by ligation of the right superior thyroid artery or thyroidectomy about one week later. If it is necessary to do a second ligation the thyroidectomy should be deferred until the patient shows the improvement that follows ligation. This is usually noticeable in from one to three months.

Conclusions. In conslusion, I would like to emphasize several points we have reviewed:

1. All diseases of the thyroid have a medical aspect, and patients having a disease of the thyroid should be placed under the observation of the internist, so that thorough clinical and laboratory studies can be made in arriving at a diagnosis before treatment is instituted.

2. Colloid goiter is a medical disease. In districts where it is endemic it should be prevented by the administration of iodin. School children in all districts up to the age of twenty years should be examined yearly and those cases showing enlargement of the thyroid should be placed on iodin, giving them a course of sodium iodid twice yearly, or one dose of sodium iodid weekly over a period of several years. Adults who have remained untreated and who have diffuse colloid adenomata should be placed on sodium iodid for a long period, and if the gland does not respond to this

treatment a double resection of the thyroid may be done and the patient should again be placed upon small doses of sodium iodid over a period of years.

3. Adenomatous goiter should be treated surgically after maturity, or before if pressure symptoms develop, or for cosmetic reasons. The surgical treatment is the enucleation of a single adenoma and resection of one or both lobes for multiple adenomata.

4. All cases of exophthalmic goiter should be under observation and treatment by the internist in preparation for surgical treatment during the time of surgical treatment and after operation. The medical treatment consists chiefly of rest, regulating the diet, maintaining the water balance and stimulating the heart. The surgical treatment is the ligation of one or both superior thyroid arteries, followed by thyroidectomy, in order to cut off the stimulation, to reduce the blood supply and to remove a large amount of the secreting glandular epithelium.

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DISCUSSION

Dr. George E. Pfahler: I am sure we all appreciate this very thorough paper that Dr. Horgan has presented to us, and I am sorry that he could not present it as completely as he had planned. I think if we all considered the physiology of these organs, and the pathology secondary, that we could understand his proposition. I think, too, we will all agree that these cases ought to be under the observation of the internist. I believe the best arrangement is that followed at the Massachusetts General Hospital, where every goiter case is reviewed by the internist, the surgeon and the radiologist before any treatment is undertaken, and the plan of treatment is then outlined by the three. The patient is observed from time to time during this course of treatment, and at any time the treatment is changed according to the indications, either directly to medical treatment or to surgery, as indicated, or radiation alone is depended upon. I think, in recent years, most of the patients have had the benefit of medical treatment, and when possible a certain amount of rest; but most of the patients have had to go about their daily duties, and this class of patients especially have been placed under radiation treatment at least for a time. Later some of these cases are operated upon.

Now, I have had the privilege of treating about 300 cases of goiter with hyperthyroidism. I prefer to treat these cases where there are definite symptoms of hyperthyroidism, because with the x-ray or radium we can reduce this secretion, as you can reduce it with excision. You can do it gradually and stop when you have reduced it enough or nearly enough. This can be controlled by a careful study of the pulse-rate and the symptoms, and by basal metabolism tests. The observation that helped me more than anything else is the study of the instability of the pulse. For instance, if you take the pulse-rate under rest, and then have the patient walk a few times across the floor, a normal person will have a rise of pulse-rate, the hyperthyroid case will have probably three times as much rise relatively. Now, as the patient improves that difference becomes less and less and approaches normal. That is a simple office test. Of course, the basal metabolism during treatment, or after treatment, gives perhaps more satisfaction and is a more accurate method,

but the other is a simple office test that may be made each time. We can also watch the progressive improvement in weight and the reduction in nervous symptoms. We can see these improvements from day to day; we see them usually at the end of the month, and from that time on they are usually progressively better. Only 4 cases encountered have had to be operated upon after radiological treatment was discarded. I think I have more than that many who required radiation treatment after operation. I had a case day before yesterday who had been operated on very thoroughly and still had hyperthyroid symptoms. We also have a number of cases of hyperthyroidism without any palpable goiter, and they, too, respond to radiation treatment. The next point I would like to emphasize is that we can treat these cases too long by radiation. We give the treatments once a month now. If the patient has not shown definite improvement within a month, and give reasonable promise of recovery within five months, then perhaps the patient had better be operated upon. That is the conclusion that the committee has come to at the Massachusetts General Hospital. A number of these patients can be made more favorable for operation if operation is desired, because you are reducing the pulse-rate and building up their general system preparatory to operation, so that during this period, as Dr. Horgan recommends, while the patient is waiting to be prepared for operation he might as well have the benefit of some radiation and reduce this secretion.

Dr. Henry P. Brown, Jr.: In view of the lateness of the hour, I will not take up much of your time. It has certainly been a pleasure and privilege to hear Dr. Horgan's paper, and I am sorry that we could not have the pleasure of his finishing it. This question of disease or malfunction of the thyroid is one which is of importance to every member of the medical profession, and any light that can be thrown upon it is most welcome. In regard to basal metabolism, I think it is destined to become one of the most important aids in the study of this disease. I think that as experience accumulates and we are able to improve our technic and interpret our results, it will be almost comparable to the position which the *x*-ray holds at present in helping to diagnose surgical conditions of the stomach. That, perhaps, may not be too optimistic when you consider the position the *x*-ray held twenty years ago and as it is now. The question of *x*-ray treatment is most interesting, and no doubt if these cases of toxic goiter could be referred early to the roentgenologist there would be more chance for improvement than with late cases. I was interested in Dr. Pfahler's remark that if he does not improve the condition in four or five months he then turns the patient over to the surgeon. During that time you have a chance for the cardiac and nervous system to become severely damaged, and it is questionable whether it is worth waiting that long. It is important to bear in mind

that the x-ray is not without its dangers. It seems to me the impression that if the x-ray cannot help it cannot hurt is wrong. This was impressed on me by the case of a woman who came into the hospital with an acute toxic goiter and was treated with the x-ray; there was a flare-up of her symptoms and the patient passed away. If this occurs only once in 1000 cases, yet if it happens to be your own patient, it makes a considerable difference; you forget about the 999. To me the most brutal and illogical method of treating toxic goiters, and yet one which in our present limited knowledge of the condition gives the best results, is the surgical treatment. It does not seem logical that just because a gland is not functioning properly we should remove three-fourths or four-fifths of it. If we had an infected foot we would not want to remove three-fourths or four-fifths. In view of our limited knowledge of disease of the thyroid and its allied endocrines, surgery seems to be the most beneficial factor. This is shown in Crile's Clinic in Cleveland, and he has reduced his mortality to under 2 per cent, which is really "going some." We realize that in this (Crile's) Clinic they have made a specialty of the study and treatment and have had many more cases than we see in this part of the country. I am sure that I express the opinion of most surgeons when I say that we are looking forward to the time when these cases of toxic goiter will be successfully treated by the internists and will be taken out of the hands of the surgeon.

Dr. Solomon Solis-Cohen (in answer to a request from the Chair): My views on goiter are familiar, or ought to be familiar, to all present, and I merely ask a question of Dr. Brown because it opens a certain important phase of the subject. Dr. Brown advocates surgery in "toxic goiter," and as to that I agree with him; but I do not agree with him if by toxic goiter he means Graves' disorder. I should like to know if that is what is in his mind.

Dr. Brown: Dr. Cohen, I do not care by what term you call it, Graves' disease, or Basedow's disease, or exophthalmic goiter, it is the clinical picture of toxic goiter. I hope that the time will come when we will not see these extreme toxic manifestations, and we will be able to recognize the early symptoms of toxic goiter as we now do that of appendicitis, and not wait until too late. It is up to you medical men to recognize this and turn these toxic cases over to the surgeon for early treatment.

Dr. Cohen: Then, Mr. President, I will accept your invitation to talk. Nomenclature is far from unimportant. Errors in nomenclature are at the bottom of two common failures: First, the failure to recognize Graves' disorder early, and second, the failure to realize that Graves'

disorder and "toxic goiter" are not the same thing, but two different and distinct pathologic entities which must be managed differently. Graves' disorder is that condition commonly and misleadingly termed "exophthalmic goiter," but which can occur without goiter and without exophthalmos. Those who follow the Teutons call it "Basedow's disease;" some, "Parry's disease." The eponyms merely indicate a difference of credit to the original observer; they do not lead astray. But, however we name it, it is not primarily a thyroid affection; the thyroid disturbance is an incident in its evolution. Toxic goiter, on the other hand—or, to follow Plummer, "toxic thyroid adenoma"—is primarily a tumor of the thyroid gland, which, by hypersecretion, gives rise to toxic symptoms. There is a great deal of difference between removing or destroying this essential toxic adenoma and removing or destroying a thyroid gland which is malfunctioning because of pathological changes originating elsewhere. Now, in Graves' syndrome the malfunction, with or without enlargement of the thyroid gland, is dependent upon disorder of the involuntary nerve system and its endocrine adnexa. I mean the whole autonomic nerve system—that which the French term the parasympathetic as well as the sympathetic; and it may be pointed out, in passing, that here again those who follow Teutonic models have introduced harmful confusion into nomenclature by attempting to restrict Langley's term "autonomic" to the craniosacral division of the involuntary nerves, when it should be applied to the whole system. I mean also that not only the thyroid gland, but also other endocrine bodies—certainly the thymus, probably the suprarenals, possibly the pituitary, sometimes the pancreas—are involved together with the autonomic nerves. Destruction of the thyroid may break a vicious circle and remove many symptoms, but it does not bring about full recovery from this complex disorder. Obviously, therefore, it is important to recognize that among the complex syndromes presented to the internist and the surgeon, and which may include thyroid tumor, tachycardia, increased basal metabolism and various nervous disturbances, there are two very distinct ailments; and if we clinch that recognition by consistently applying to these two distinct ailments two distinct names, we are likely to manage our cases better than if we confuse the two conditions under one name. Such confusion is especially dangerous if the name adopted be "toxic goiter," for then we should naturally submit all our patients to surgery; and apart from the risk of operative death, surgery destroys in large part an organ necessary to the balance of function. That this is most frequently, if not always, necessary, in dealing with an essential thyroid tumor, that by reason of its excessive secretory activity is poisoning the patient is true; for, at present, we know no certain means of healing it, and must therefore destroy enough tissue to get rid of the intoxication. We may destroy by radium or by

x-ray; we may destroy by cautery, by electrolysis, by the knife; we may destroy by ligation, to cut off nutrition; but whatever be the destructive means employed, it is surgery and not medicine, because medicine saves while surgery destroys. In Graves' disorder, on the contrary, we do know how to remedy the malfunction of the thyroid gland, and to an extent the underlying aberration of the autonomic system, by medicinal, hygienic, physical and psychic measures. In other words, the gland can be saved for its future contribution to the normal life of the individual. We can save it by doing what Dr. Foster urges us to do in nephritis, namely, by making an early diagnosis. Once in a while one puts things in a way that he thinks good, and he is prone to repeat the saying. That is my excuse for repeating the answer I made to a question put by a member of the New York State Medical Society, several years ago, in the discussion of my address before that body on "The Non-surgical Management of Graves' Disorder." The query was, "What would you consider an indication for operative treatment?" and the reply was, "A belated diagnosis." By this I mean that if the case has gone so far that the enlarged thyroid has become a danger mechanically, or has become so poisonous that there is great danger in waiting for its restoration to normal function, then, of course, we have Hobson's choice. It is true that surgeons urge operation in early cases to prevent these dangers and that they are not eager to operate upon far-advanced cases; but the early cases do not need operation. They can all recover without it; some with a minimum of medical assistance. As to the advanced cases, I should myself prefer some other method than exsection, if available. But Graves' disorder ought to be recognized early. Sometimes the problem is difficult, but commonly it is one of the easiest things in the world if the physician be on the alert. My colleague, Dr. Bram, recently presented here an excellent paper on this topic, and it is fairly well treated in the text-books, so we need not occupy time therewith tonight. I may say, however, that if instead of calling syndromes that we do not at once understand "neurasthenia," "tuberculosis," "nervous dyspepsia," "hysteria," "dysmenorrhea," "disordered action of the heart," and so on and so forth, we but take time to analyze our cases and to learn the morbid physiology that we are dealing with, we should recognize Graves' disorder early enough to assure recovery without surgical procedure. Moreover, the rest, mental and physical, that is the keystone of the management of early Graves' disorder will harm no one. It will indeed be of benefit in most of the nervous and cardiac cases, and during its progress give opportunity for closer study. To suspect Graves' disorder and treat for it, in a case of neurasthenia or hysteria, or even one of acute tuberculosis, will therefore be much less harmful than to make the opposite error. With care we can avoid both. But to repeat what I said in the beginning, correct nomenclautre is necessary to straight thinking.

Dr. Horgan (closing): I wish to thank the gentlemen who were kind enough to discuss my paper. One of the points that I wanted to make clear in the paper is that each case of goiter under observation should be studied very carefully, to determine the variety of goiter before anything is done in the way of treatment, and I think that all thyroid cases should be handled from the beginning by an internist, and if the case is a surgical one, that the medical associate follow it and treat it conjointly with the surgeon.

The paper dealt chiefly with the common varieties of goiter, namely, colloid, adenomatous and exophthalmic goiter. Colloid goiter is a disease which is endemic in certain regions, but it is found in almost every part of the country. It is more common in the so-called goitrous regions. The disease can be cured and should be prevented. I believe that if we carefully watch school children, may cases of goiter could be found early and cured, so that trouble later in life could be avoided. This is one of the things I wanted to stress in the paper. Another thing that I wish to stress with regard to colloid goiter is that it is a medical disease. Colloid goiter should never be operated upon unless the case is a long-standing one, with colloid adenomata throughout the gland which will not respond to medical treatment. Furthermore, colloid goiter should not be treated by *x*-ray.

In discussing adenomatous goiter, I tried to show that there is a hyperthyroidism, found in and characteristic of adenomatous goiter, which is insidious in its onset and which may not produce detectable symptoms for a number of years. Occasionally we see a hyperthyroidism in an adenoma of the thyroid which is similar to the hyperthyroidism of exophthalmic goiter. This condition is found in the cases in which there is active cellular regeneration within the adenoma. In some of these cases the pathological study reveals well-developed acini with hypertrophy of the acinic cells and the microscopic appearance of the epithelial cells is strikingly similar to the cellular findings in exophthalmic goiter. It must also be remembered that we may have exophthalmic goiter in a patient having an adenoma of the thyroid. In such a case the adenoma is not producing the disease and the pathological study will reveal hypertrophy of the acinic cells throughout the parenchyma. Adenomatous goiter has only been treated by Means and Holmes in a few cases where the patient refused operation. They do not advise *x*-ray as a treatment of adenomatous goiter.

Exophthalmic goiter is a disease the cause of which we do not know, nor do we know the chemistry of the secretion that is elaborated in this disease. I think that the best method of treatment is surgical, and it is accomplished by, first, stopping the stimulation; second, reducing the blood supply; and third, removing a large amount of the secreting glandular epithelium.

PROCEEDINGS

OF THE

SECTION ON MEDICAL HISTORY

MARCH 29

A Physician in the Papal Chair

BY DAVID RIESMAN, M.D.

A FEW years ago, while reading Sedgwick's *Italy in the Thirteenth Century,* I found it stated there that one of the popes had been a physician. This intrigued me and I corresponded with Mr. Sedgwick, and eventually began a bit of research in the hope of learning something of the man who, starting from the modest rank of doctor, had risen to the highest elective office in the world. In our day the loftiest office attained by one of the medical profession was that of the premiership of France (held in the most critical time in all history by Georges Clemenceau). The British Ambassador to the United States, who fills a distinguished post with distinction, is also a doctor.

I have said that the office of pope was the highest elective position in the world; I ought to say that in the age of which I am speaking the leadership of the Holy Roman Empire was also elective, the office not becoming hereditary until centuries later. Inasmuch as the German Emperor in the person of Henry IV had gone to Canossa and knelt penitently at the feet of Hildebrand, Gregory VII, Christendom had come to look upon the Pope as suzerain and overlord of all temporary rulers.

Let us see now who this man was that exchanged the doctor's

hood for the papal tiara, the waterglass and scalpel for the ring and keys. For a better understanding of the subject it is necessary to delve a little into the thrilling story of that remarkable thirteenth century, to some scholars the greatest of all since time began. According to taste, scholarship and dogma, men differ in their views as to the preëminence of centuries. Professor Braisted, the Egyptologist, thinks that the twenty-ninth century B.C. was the greatest. I wonder what he will say when he returns from Egypt, where he now is with Lord Carnarvon at the tomb of Tutenkhamen? Paul Shorey and Gilbert Murray would probably choose the fifth century B.C.; J. J. Walsh would put none above the thirteenth.* Many in whom the spirit of the Renaissance has entered contend that the palm belongs to the quattrocento. I shall not stop to discuss the relative merits of these varied predilections but shall content myself with saying that the thirteenth century was a wonderful epoch, a time of intellectual awakening, and, further, that by preparing the way for the Renaissance it has high claim to our admiration.

In the political field important things happened in that century. It witnessed the gigantic struggle between the papacy and the temporal sovereigns of the Holy Roman Empire. Frederick II, King of Sicily, was Emperor. He looms against the background of his epoch as a colossal figure, valiant, brilliant, liberal, progressive—Stupor Mundi his contemporaries called him. Descended from Frederick Barbarossa, the Hohenstaufen, on his father's side, and from the Normans on his mother's, the blood of two domineering races were so mixed in him that he could not if he would accept, as his predecessors had done, the suzerainty of the bishops of Rome. Had not his grandfather, Frederick Barbarossa, knelt down to permit Alexander III to place his foot upon his neck?

Frederick's Court at Palermo was the seat of the highest culture of the time. All races were tolerated; all languages were spoken there. To the great disgust of the orthodox, Frederick received

* Funck-Brentans, in a recent work on the middle ages, states that for France the eleventh is the greatest century.

and respected the Saracens, and even adopted some of their customs. "When he realized how each great religion reviled the other, he concluded," says H. G. Wells,[1] "that all religions were impostors." Thus, by his doctrinal heresy on the one hand and his political independence on the other, he brought upon himself the undying hatred of the Church. To medical men he is interesting because he laid down in precise fashion a five-year curriculum for the education of physicians at the University of Salerno.[2]

There is a certain analogy between Frederick II, who was of Swabian descent, and the last of the Hohenzollerns, also of Swabian extraction, now a resident of Holland. Both concerned themselves with everything under the sun, and there is a further resemblance in that both at the height of their glory undertook the impossible and failed utterly.

Frederick's conflict with the popes is a sad and bitter tale; a tale of heroic courage on the part of the emperor and of unbelievable perfidy on the part of emperor and pope. In the end Frederick succumbed to that irresistible papal weapon, excommunication, hurled against him by Innocent IV at the Council of Lyons. His admirable son Manfred and his grandson Conradin took up the hereditary fight, but in vain, for the Pope had now won a great ally, the sinister, unrelenting Charles of Anjou, in somberness, in intolerance, in vindictiveness, the prototype of Philip II of Spain. With Conradin perished forever the masterful and attractive race of the Hohenstaufens.

As soon as Charles of Anjou became King of Sicily he at once reached out to get control of the papacy, and when Pope Gregory X died on January 12, 1276, Charles succeeded in procuring the election of a Frenchman, Peter of Tarentaise, a pupil of the great Thomas Aquinas. This Peter, who took the name of Innocent V, was the first Dominican to become pope. Although a great scholar he was a weakling, and became the ready tool of

[1] Outlines of History, New York, 1921.

[2] Allshorn, Lionel: The Life and Times of Frederick II, Emperor of the Romans, King of Sicily and Jerusalem, 1194–1250. "If a temple should ever be raised to the memory of those who have struggled for the freedom of man, then Frederick should find a high place in the sanctuary."

Charles to the detriment of the influence of the papacy and of the freedom of Italy. He endowed Charles with great political power, making him senator of Rome and vicar in Tuscany. The Roman climate, however, was too much for Innocent V—he succumbed six months after attaining the Chair of St. Peter.

Unfortunately, the second Council of Lyons had entrusted Charles of Anjou with supervision of the conclave. Charles was naturally eager for the election of another French pope, and with that end in view treated the Italian cardinals who formed the majority with extreme rigor, carrying out literally the decrees of the Lyons Council. The cardinals were locked in; their food was restricted to wine and bread, and, although it was July, all the windows and openings were walled up so that, writes Suba Malaspina, not even a bird could have penetrated into the conclave. Nevertheless, Charles failed. The cardinals chose an Italian, Ottoboni Fiesco, Adrian V, as pope. Poor Adrian, however, had suffered so much through Charles' cruel treatment that he died at Viterbo on August 18, 1276, thirty-eight days after his election.

A new conclave was assembled at once. Fortunately for the much-tried cardinals, chiefly men of advanced age, Adrian a few days after his election had rescinded the decrees of the Lyons Council regarding conclaves. The cardinals waited more than the stipulated ten days in order to give the absent members of the college time to arrive. This delay and the inability to reach a rapid decision prolonged the conclave to a point beyond the endurance of the excitable Viterbians. They assaulted the emissaries of the conclave and made much noise and disturbance. Under such pressure the cardinals reached a unanimous decision sooner than they had intended and elected, somewhere between the 13th and 20th of September, 1276, the Cardinal Bishop of Tusculum, Petrus Hispanus, a Portuguese physician, as Pope John XXI. Of him this essay treats.

The choice really should have fallen on the great Cardinal Deacon John Cajetanus Orsini; but the conclave was afraid of offending Charles of Anjou by electing an Italian, a Frenchman they would not choose, so they picked a man from the neutral

country of Portugal. John Orsini had, however, not long to wait. He soon succeeded the man whose election he himself, with a remarkable degree of self-effacement, had brought about. As Nicholas III he followed John XXI in the Chair in 1277.

To return to John XXI. This Pope was before his election not only a high church dignitary but also a physician. His name, Petrus Hispanus, was a medical household word in the Middle Ages. He is the only one of the profession that has ever sat in the Chair of St. Peter. Some writers, it is true, speak of another Pope as having been a physician, Paul II (Pietro Barbo). Paul, however, never studied medicine. The legend, if so it can be called, arose from the fact, as I found in the delightful chronicles of Platina, that Paul like many laymen was fond of prescribing for his friends. When any of the cardinals or anyone of his personal entourage was ill, he would send draughts and potions to the helpless patient. In that way the belief arose that Paul was a doctor of medicine.

Something ought to be said in the beginning regarding Petrus' papal name John XXI. According to nearly all writers, he should have been called John XX, as there were apparently only nineteen Johns before him. On one of his seals he is designated as John XX and in his epitaph as John XXI.[3]

Various explanations have been given for the discrepancy. It seems that the pontificate of John XIV was by some chroniclers divided into two parts, the one preceding and the other following his imprisonment in Castle Sant' Angelo. In that way he is both John XIV and John XV.

Another explanation is that the anti-pope of Gregory V was designated (by John of Piacenza) as John XVII.

A third explanation is the one given by Gregorovius, namely, that the belief in a Popess Joanna influenced the enumeration. It

[3] An amusing reference to this discrepancy is found in J. B. Cabell's Jurgen (New York, 1921). The hero in order to get into Heaven from Hell pretends to be Pope John XX and uses the "Cantrap" given to him by the "Master philologist," namely: "At the death of Hadrian V, Pedro Juliani, who should be named John XX, was through an error in the reckoning elevated to the papal chair as John XXI."

may not be without interest to give a brief account of this legend. A woman, in order not to be separated from her lover who was a monk, entered a Benedictine monastery under the name of John Anglus. Later she accompanied her lover to Athens, acquired the Greek language, and afterwards returned to Rome, where she soon gained great fame for her learning and piety. Eventually, from professor of Greek she was raised to the papacy, there being no suspicion of her sex. She exercised the power of the Church to the admiration and respect of all. Meanwhile she became suddenly overwhelmed by an unmistakable intimation that her secret relations with the monk were about to bear fruit. An angel appeared to her one night as her term approached and announced to her that she might choose one of two alternatives: either to hide her condition, suffer in secret, conceal her offspring, continue to rule the Church, and finally lose her soul; or to endure the unspeakable humiliation and disgrace of a public disclosure, be overwhelmed with infamy, be thrust from the Church, and in the end be admitted to Heaven. The poor woman had a great and noble soul. She chose the better part, and in token that her expiation was acceptable to Heaven it was granted to her to become a mother while the public procession in which she took part was returning to the basilica of St. John Lateran.[4] [5]

This remarkable legend has of course no basis in fact. The chroniclers living at the time of John XXI do not speak of it; so that either it sprang up afterward or they did not consider it of sufficient importance to mention it in their chronicles.

In the magnificent church of San Paolo fuori le Mura in Rome there are medallion portraits of all the popes since St. Peter. To my surprise I found twenty Johns preceding our John XXI. As no dates are given on the medallions, only the length of reign, it was not possible to tell precisely the time in which the various Johns lived. The Church evidently, unlike the historians, sees no discrepancy in the Johannesian enumeration.

[4] Chisholm: Recreations of a Physician.

[5] Rhodis, P. J.: Leipzig, 1904. A biting satire in which the father of Popess Joanna is an English monk, a pupil of Scotus Erigena, and the mother a Saxon goose-girl named Jutta.

Petrus Hispanus, Pope John XXI, was a native of Portugal. The name Hispanus was due to the fact that in his age little distinction was made between Portugal and Spain. He was born near the beginning of the second decade of the thirteenth century, the son of Julianus, a physician. Little is known of his early life. He first appears as a student in the University of Paris, where as a fellow-pupil of Roger Bacon he came under the influence of the great English logician, William Shyreswood. The latter must have been a man of extraordinary attainments, for Roger Bacon ranks him above Albertus Magnus, the dominant scholastic of his time.

As medicine was not then sharply separated from the other branches of learning, it was not a great step for Petrus Hispanus to pass from logic, including as it did Aristotle and the Arabian philosophers, to medicine. Some believe that Petrus studied logic as well as theology at Paris and medicine at the more famous faculty of Montpellier. It seems more probable, however, that he was made *magister* of medicine at Paris.

The University of Paris at this time was passing through a period of reaction. The reading of Aristotle in public as well as in private was forbidden.[6]

It would be interesting if we could, through a manuscript of Roger Bacon's,[7] get a sidelight on the life of his fellow-pupil, the subsequent Pope. Intellectually, Bacon was infinitely greater than Petrus. Far in advance of his time, he insisted that the renewal of the sciences could be brought about only by a return to investigation of original sources: In the case of philology to the Greeks, of nature to observation, and of theology to the Bible. The foundation of natural philosophy, he insisted, was mathematics; the only way to a knowledge of nature, experiment. Small wonder that these ideas brought Bacon into conflict with the Church and led to his persecution and eventually to his imprisonment at the hands of the ecclesiastical authorities.

[6] The University of Toulouse, on the other hand, by direct edict permitted the study of Aristotle, and prided itself on that fact.

[7] The original MS. which Professor Newbold is engaged in deciphering

At some time in his career, whether at Paris or elsewhere is not known, Petrus Hispanus wrote a book on logic called *Summulae logicales*.[8] There has been much dispute about the author of this book, which is an adaptation of one written by Petrus' teacher, William Shyreswood. Some have attributed the authorship to Petrus Hispanus, a Dominican, others to Michael Psellus the younger. Owing to the meagerness of transmitted data the identification of medieval personages is often difficult; this is especially true in the case of Petrus Hispanus, since that name occurs with great frequency in the literature of the Middle Ages. Thus the single order of Dominicans counts no less than six men of the name of Petrus Hispanus, for one of whom they have claimed the honor of having written the *Summulae logicales*. But since monkish writers are always known as *fratres*, never as *magistri*, and as Petrus Hispanus is always called *magister* and never *frater*, he could not have been a member of a monkish order. Thus Ricobaldus of Ferrara, Canon of Ravenna,[9] wrote in 1312: *Hic* (*i. e.*, John XXI) *magnus magister . . . cui nomen fuit magister Petrus Hispanus, qui tractatus in logica composuit.* Moreover, as Pope, Petrus Hispanus showed himself inimical to the monks, particularly to the Dominicans. In view of these facts prominent modern Dominicans[10] admit that Petrus Hispanus was not one of their sect.[11]

In 1597 a Greek MS. was discovered in Augsburg (it is now in Munich), the text of which corresponds almost word for word with the *Summulae logicales* of Petrus Hispanus. As author of the Greek work the title names Michael Psellus, a Byzantine writer who lived between 1018 and 1079, nearly two hundred years before Petrus. This discovery seemed to prove that the

[8] Franck, A.: Dict. des Sc. philosoph., Paris, 1875, p. 1333. John XXI is called one of the most celebrated logicians of the school of Paris.

[9] Quoted by Stapper: Papst Johannes XXI, Munster *i. w.*, 1898, p. 14.

[10] Quétif-Echard, Scriptores ord. Praed., i, 485 *et seq.*, Denifle, quoted by Stapper Pabst Johannes, xxi, Muenster, i, iv., 1898.

[11] In the "Monumenta Germanica," in three places, the names of Petrus Hispanus and John XXI are used synonymously; once the term Petrus Phisicus is employed; once John XXI; once John XX. In practically all the MSS. of the fifteenth and sixteenth centuries, Petrus Hispanus, the logician and physician, and John XXI are identified as one.

Summulae logicales of Petrus Hispanus was nothing more than a copy from a Greek original. But both by internal evidence and by the discovery of numerous other MSS., it has been demonstrated that the synopsis of Michael Psellus is a Greek translation made in the fifteenth century by Georgios Scholarios from the *Summulae logicales* of Petrus Hispanus.

The Pope has also been confused with a philosopher logician, Petrus Hispanus. Nicolao Antonio[12] has, however, straightened out this difficulty by showing that the logician was not even a contemporary of the Pope. He was professor at Paris (perhaps also at Siena) and a Dominican. Another Petrus Hispanus, at times confounded with our Pope, was professor of canonical law at Bologna. He likewise was not an actual contemporary, being engaged in teaching as early as 1223.

The *Summulae logicales* enjoyed great popularity throughout the Middle Ages and later was frequently reprinted, and always as the work of Pope John XXI. Prantl found no less than forty-eight imprints made in the first century after the invention of printing.

It is to this work that Dante refers in his *Divina Commedia*. Meeting John in the seventh circle of glorified souls, Dante's guide, the Franciscan Bonaventura, points out Petrus Hispanus, saying:

E Pietro Ispano

Lo qual giu luce in dodici libelli[13] ("And he of Spain in his twelve volumes shining").[14]

John XXI is the only one of the contemporary popes whom Dante meets in Paradise.

About the year 1247 we find Petrus Hispanus as a teacher of medicine at Siena when that ambitious town was about to establish a university of its own. It was there that he wrote his first medical work, *A Dietetic Treatment of Surgical Patients*, at the request of his colleague, the surgeon, John Mordentis of Faenza.

[12] Quoted by Henschel, A. W. E. J.: Janus, 1851–1853, i–ii, 526.
[13] Canto XII, v, 135–136.
[14] Cary's translation, New York, 1897.

There is also a record in the archives of Siena of a stipend paid to him for services to the community.

Being a native of Portugal, where at that time the Moors were still a power, he probably understood Arabic, which readily accounts for the fact that we have from his pen commentaries on Avicenna and Isaac Judaeus. Commentaries on Hippocrates and on Galen are also attributed to him. His greatest medical work, however, is the *Thesaurus pauperum* (the Treasury or Pharmacopeia of the Poor). This famous treatise, printed for the first time in Antwerp in 1476, was composed either in Italian or in Latin, and was in all probability written at the Court of Gregory X, to whom *qui pater pauperum nuncupatur*, it is dedicated.

Because printed in an early Spanish edition, some (Haeser) have attributed the work to Petrus Julianus, the father of the Pope; but the Spanish is a translation, not the original. Moreover, all references by the medieval chroniclers are against such a view. In the *Practica* of Serapion, printed in 1525, occurs the following sentence: "Here begins the *Thesaurus pauperum* of John XX, pontifex maximus, whose name formerly was Petrus Hyspanus: In which thou findest absolutely the theory and the practice of curing diseases."

There is a striking similarity in style between the *Summulae logicales* and the *Thesaurus pauperum*. Platina,[15] the archivist of Leo X, says: "He wrote many Tracts in his life, especially certain Rules relating to Physick, for he was counted a very good Physician. He wrote also another book and called it *Thesaurus pauperum*, or the Poor's Treasure, and set out Problems in imitation of Aristotle." The *Thesaurus* is a collection of recipes for every disease under the sun. It had an enormous vogue in the Middle Ages. Being copied and recopied before the discovery of printing, every copyist added his own favorite prescriptions until the work finally became a hodge-podge of medical treatment and magical formulas of the most grotesque sort.[16]

[15] Platina, Baptista: Lives of the Popes, London, 1685.

[16] The Thesaurus pauperum of Petrus Hispanus must not be confounded with a work of the same title written a little later by Arnald of Villanova.

In addition to the *Thesaurus pauperum*, John composed another medically famous work, the *Liber de oculo* (Book on Diseases of the Eye). First printed in 1476, the *Liber de oculo*, which has recently been edited by Dr. A. M. Berger of Munich, enjoyed a high degree of popularity for several centuries. The great Guy de Chauliac, in his *Chirurgia*, refers to it, and Michelangelo copied its prescriptions for his own use. In the Vatican Library I saw this precious document in the handwriting of the great Florentine. Arnald of Villanova, in his *Breviarium*, also reproduces some of the remedies and prescriptions, especially the famous *aqua mirabilis*. This "wonder water" of Petrus Hispanus consisted of filings of silver, copper, gold, etc., in a boy's urine. "It elevates the physician who can prepare it to the rank of prophet."

Judged by our standards, the *Liber de oculo* is of little scientific value, but will not the physicians of seven hundred years hence be likely to say that of many of our own works?

Petrus Hispanus places the seat of vision in the lens, which is of course false, since the lens can be removed, as in the operation for cataract, without destroying vision. Vision is brought about by a *spiritus visibilis* or visual ether. In this hypothesis, adopted from Empedocles and Galen, Petrus departs from the theories of Aristotle, who had assumed the motion of some medium between the eye and the object, and had thus laid the foundation of the undulatory theory of light.

The first to form a clear notion of light rays in the eye was Kepler (died 1631), who held that an inverted image was produced on the retina.

Petrus Hispanus deserves great credit for one fact in particular —namely, he tried his best to exclude superstitious and sympathetic remedies from his work,[17] which is the more noteworthy, as the somewhat later Arnald of Villanova, already mentioned, includes in his book remedies against demons and evil spirits.

But with all his skepticism, Petrus was a child of his time and believed firmly in astrology, holding that the human body was

[17] "Redeamus ad naturalia," he writes.

under the influence of planets and zodiacal signs. In this respect the great medieval philosopher and physician, Moses Maimonides, who preceded him by seventy-five years, was far in advance, for he wrote as follows: "Man should only believe what he can grasp with his intellectual faculties, or perceive by his senses, or what he can accept on trustworthy authority. Beyond this nothing should be believed. Astrological statements, not being founded on any of these sources of knowledge, must be rejected."

I have as yet said nothing of Petrus' positions and experiences in the Church, about which we must be informed in order to understand his transfer from the ranks of physician to the highest pinnacle in the Roman hierarchy.

His first ecclesiastic position was that of canon of Leon. Soon after receiving this appointment he attracted the attention of Cardinal Ottoboni Fiesco, later Adrian V, and accompanied him as personal physician on his journeys as papal legate. About the same time he was made deacon of Lisbon with the unusual privilege, testifying to his importance, of non-residence. In the service of Cardinal Ottoboni he came into contact with Cardinal Thedaldus Visconti, and when that dignitary became Pope Gregory X, he made Petrus Hispanus his body physician or archiater. From now on Petrus' ecclesiastic preferment was rapid. He was elected Archbishop of Braga in 1273 and in the same year Cardinal Bishop of Tusculum. His consecration as cardinal took place in 1273 at the Second Council of Lyons, simultaneously with that of the lovable Bonaventura the Minorite and of Peter of Tarentaise the Dominican. Medieval historians make much of this triple consecration.

After ascending the papal chair, one of John's first acts was to confirm his predecessor's edict suspending the Lyons Decrees regarding conclaves. His enemies criticized him for this audacity in rescinding a decree of an Ecumenical Council and had a certain measure of right on their side in doing so. They remembered that prior to the election of Pope Gregory X, in 1271, the Holy See had been vacant for three years because the wrangling cardinals could not agree upon a pope.

Pope John was most approachable, particularly to poor scholars. It was this trait in his character that made him unpopular with the higher clergy, who contended that such conduct lowered the dignity of the papacy. The ignorant chroniclers speak of him as "learned and silly; a wise fool upon the sacred chair; a man without presence or dignity, who loved learning and hated monks." Platina[18] writes in a similar vein: "He did many things that argued him to be guilty of folly and levity, and does not deserve commendation unless it be for one thing, and that was that he assisted young scholars, especially the poorer sort, with money and preferment."

Some of the calumnies against the Pope can be traced to the Dominicans, whose special enmity he seems to have incurred. As I have already stated, John never belonged to any of the monkish orders and was a *magister* and not a *frater*. His knowledge of medicine and astrology in their eyes bordered on magic; as one of them wrote: "*Joannes Papa magus, in omnibus disciplinis instructus, religiosis infestus.*" If John showed any preference for any of the monks it was for the Franciscans, the Minorites. This was due partly to the fact that his sponsor at the conclave that elected him, John Cajetanus Orsini, was the Protector of the Franciscan order, and partly to the other fact that the principles of poverty and humility of the order of St. Francis appealed to him. He had determined, as proof of his good will, to make John of Parma, general of the order of the Minorites, a cardinal, but died before he could carry out his plan.

The Dominicans and the Franciscans hated each other with a most unclerical hatred (or perhaps clerical?). Even the beatific Fra Angelico, whose pictures prove him to have been one of the sweetest souls of the Renaissance, was not free from anti-Franciscan rancour. He painted Christ being welcomed by Dominicans "as only Dominicans can welcome Christ," and in his painting of the "Last Judgment," he sends the Dominicans to Heaven and the Franciscans to Hell.

[18] Loc. cit.

In his political relations John XXI showed strength and independence; much more indeed than he is credited with by some of the later writers.[19] He first of all insisted that Charles of Anjou should renew his previous oath that he would not combine the crown of Sicily with that of the Roman Empire or of Lombardy. With the recently elected German Emperor, Rudolph of Hapsburg, who ascended the throne in 1273, after an interregnum of twenty-two years, he carried on negotiations in which he insisted, against Rudolph, upon his rights in the papal states, the suzerainty of which the Emperor claimed for himself.

One of his greatest problems was the collection of the tithes. The Council of Lyons, in order to raise money for another crusade, had provided that one-tenth of the income of the churches should be sent to Rome. It had also ordered a revaluation of the ecclesiastical properties as a basis of the new taxes. Scotland was the first to rebel against the imposition of the tithes, at a convention of the clergy held at Perth in 1275. Soon afterward England began to murmur. The English clergy did not object to the taxes so much as to the new valuation. They wanted to retain the old, more liberal valuation established in 1263 by the Bishop of Norwich. To bring the matter to a climax, they sent a delegation to John XXI, complaining, among other things, that the official tax collectors lived luxuriously in London while low clerics did the collecting for them; further, that taxes had been imposed on leproseries, poor-houses and hospitals.

In a solemn consistory at Viterbo the Pope heard the complaints of the English delegation. He agreed to all they asked except to the matter of valuation. The basis of the Bishop of Norwich, he insisted, could no longer remain in force.

John had much trouble with the ruling kings and princes, who, under promise of embarking upon a crusade, had asked that the tithes might be allocated to them so that they could equip their armies. Philip III, The Bold, son of St. Louis who had led the previous crusade, swore in the presence of all his nobles

[19] Platina, Henschel, Milman and others. Some maintain that he entrusted the cares of government largely to Cardinal Orsini, subsequently Pope Nicholas III.

to lead an army against the infidels. Through the Pope's faith in his protestations the king was able to secure large sums with which he organized a great army; but instead of leading it against the Saracens, he made war against another Christian prince, Alfonso of Castile and Leon. One and all thus broke their solemn promises. They pocketed the tithes and forgot about the Holy Sepulchre.

An historically interesting phase of the Pope's activities was his negotiations with the Greeks for a union of the Eastern and Western Churches. Michael Paleologus had sent delegates to the Second Council of Lyons for the avowed purpose of bringing about a formal union with the Roman Church. In the name of Michael and of his son and heir, the Greek delegate swore to accept the Latin *symbolum*. But he swore verbally and possessed no written authorization from his sovereign to show to the Council. Soon it became clear to the leaders of the Roman Hierarchy that the Greek emperor was shamming adhesion to the Pope for the sole purpose of enlisting his help against the wily Charles of Anjou, who was preparing to make war on Michael Paleologus on behalf of the deposed Latin emperor, Baldwin II.

In order to test Michael's sincerity and to bring about an actual union of the churches, John sent delegates to Constantinople with most explicit instructions. They were to make the Greek emperor, his son and the Greek clergy swear publicly to accept the Roman doctrine and to make as many manuscript copies of the proceedings as possible. Should it not be feasible to bring about such wholesale public conversion, the delegates were to attempt conversion nevertheless, even if but few of the clergy were present. If the clergy objected to swear obedience and reverence to the Pope, the delegates were to yield this point for the time being in the expectation that the priests would acquiesce at another time. Nothing, however, came of these negotiations, for the Greek emperor and his clergy had no thought of submitting to the Roman Pontiff.

Another opportunity for union presented itself under John

Paleologus and Pope Eugenius IV, at the Council of Florence, in 1439, just before the fall of Constantinople. Although the union was actually decreed, nothing ever came of it; hence the Eastern or Greek Church and the Western or Roman Church are separate and distinct today.

Pope John had another contact with the East, curious and not cleared up entirely. The Tartar Khan Abaga sent delegates to John to inquire when the next Council would take place and promising his help against the Saracens. The delegates reported that Kublai Khan, an uncle of their chief, had embraced Christianity, and that both uncle and nephew were eager to have missionaries sent to their country.

Enthusiasm ran high at this prospect of a great accession to the Pope's spiritual dominions. After having been feted at the Papal See, the delegates were sent to the Court of France, where they were well received and lavishingly entertained until the Easter Festival of 1277, then the discovery was made that the delegates were Nestorian Christians and not Romanists. From that moment their mission became a hopeless failure.

While these more or less external questions were pressing upon the Pope, he was called upon to take action with regard to certain heretical doctrines that were spreading from the universities of Paris and Oxford and were rapidly making converts. He was busily engaged in the study of these problems when a sudden and mysterious accident cut short his career. Just behind the papal palace at Viterbo, erected on an old Temple of Hercules, John had built himself a study chamber. On May 14, 1277, the Pope had gone into this room, which had just been finished, when suddenly the ceiling crashed down upon him and crushed him. He died six days later.

His enemies, who had disapproved of his learning, propagated the legend that the Pope had been engaged in black magic and had just given his soul to the Devil when the ceiling fell. Others, however, point out that he died *munitus sacramentis ecclesiasticis*. He was buried at Viterbo in the Church of San Lorenzo and rested there forgotten until 1886, when, at the cost of the Portu-

guese Ambassador at the Holy See and of Pope Leo XIII, a new monument was erected to his memory, which bears the inscription: "*Maxima lux,*" of Portugal.

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APRIL 11

Early Anatomical Fugitive Sheets

BY LE ROY CRUMMER, M.D.

Poster advertising reached its culmination during the Great War. The beautiful design and general attractiveness of these posters led to the collector's mania, and many of these evanescent

prints now repose in the cabinets of enthusiastic amateurs. But this period did not mark a new development of poster design, since advertising by means of a single printed sheet is as old as printing itself. In fact, the very earliest impressions of the presses were almost equally distributed between single-sheet proclamations and almanacs, both of which survive with an uninterrupted history to the present.

Early proclamations, both ecclesiastical and governmental, were issued in poster form, and the presence of bulletin boards in our college halls and the nail-studded tree at the rural four corners is a relic of a custom as old as the printed page. Many a political campaign was conducted in England by means of broadsides and many a minor author achieved local distinction by means of a poem printed upon a single sheet. Artists and engravers also used this means of expression, and even some of the most celebrated of the artists and engravers of the Renaissance made plates of poems and fables of such excellence and beauty that these prints are a delight even today. The trade card and the price list, with and without illustrations, are also found among the early fugitive sheets.

The art of illustration which had become an important part of manuscript-writing was rather slow in finding a place in the printed page, but an occasional book, even in the seventh decade of the fifteenth century, contained wood-cuts. It was not until 1480, however, that they were freely used, but soon after this date, wood-cuts in books on medicine illustrated more or less anatomical detail. Among the very first of this type was the bleeding manikin, which was used both as a book illustration and as a fugitive sheet. A serial study of this single figure would show the entire history of the art of illustrating and the art of bleeding up to the time of Louis. The Zodiac man, developed entirely from the astrological charts, survives in the almanacs of today, and a combination of the Zodiac man and the bleeding manikin was frequently used in the purgation calendars of the Renaissance. The graphic anatomical incunabula represent an important epoch in the development of printing and of anatomy.

The combination of letter-press and seals or emblems in the form of broadsides was soon followed by the single sheet, with the wood-cut predominating.

The medical profession was not slow to adopt this form of publicity, and many of these sheets are of the greatest interest in the study of the historical and sociological development of medicine. The majority of such printed broadsides then, as now, pertained to the irregular by-products of medicine, and the appeal was to the less enlightened of the public in the form of illustrations of myths, monsters and the brilliant results of operations. Hollander has shown that many of these monster sheets are a source of information concerning the development of medical tenets of the time, more especially as denoting the spread of knowledge of medicine among the people.

Included in the very rare impressions of the early presses were fugitive sheets representing human anatomy, and as such sheets were evanescent and easily destroyed, very few have survived. The few that are still accessible present a phase of the growth of anatomy and anatomical illustrations that is not without interest. Probably the earliest known fugitive sheets showing definite anatomical details are the skeletons distinguished by the names of the artists, the Hela and Wechtlin skeletons. These two figures, originating as broadsides, were also frequently used, more or less modified, as book illustrations at a slightly later period. These skeleton figures appeared in 1493 and 1517. A very few sheets of varying types may be assigned to the period from 1520 to 1538, but in the period from 1538 to 1580 fugitive sheets are comparatively common. Anatomically, artistically and even commercially these fugitive sheets reached the height of their development in the figures of Remellin, 1613, but imitations of Remellin were issued as late as 1683.

The literature concerning fugitive sheets is extremely meager. Choulant[1] gives a short discussion and a rather extended check list of the known sheets in his celebrated work, but even as cor-

[1] Geschichte der Anatomischen Abbildung, Leipzig, 1852.

rected by Mortimer Frank, 1920, it is by no means complete, and his inclusive dates are erroneous in that he makes no mention of any of the sheets, except the skeleton, appearing before 1538 or after 1590. Sudhoff[2] has given us a careful analysis of the development of the Hela skeleton. Weindler[3] has been particularly interested in fugitive sheets from the gynecological standpoint, and gives illustrations of three of the Eve plates. Hollander[4] also devotes a short chapter to the anatomical fugitive sheets in his latest book.

Several of these sheets fell into my hands almost by accident, and so aroused my interest that during the year 1922 I made a careful inquiry for them in many museums and libraries of Europe. There are only two in the British Museum: one, the Wechtlin skeleton, was indexed and catalogued as a fugitive sheet, and the other, a very large sheeting, showing the Hela skeleton, was unknown until my inquiry uncovered it, carefully folded in a copy of Magnus Hundt, 1501. In the Wellcome Historical Museum there are two—the male and female C. B. copperplates, with the letter-press in English and with several superimposed flaps. These are found pasted inside the covers of a fourteenth century manuscript, and can definitely be dated about 1550. I could find none in the libraries of the College of Surgeons or the College of Physicians, and there are none in the Bodleian Library. I was unable to find any in Paris, although I suspect that this was perhaps due to my bad French rather than to a total absence of such sheets. A search which I made in the Bibliothèque Nationale, both in the Library and the Bureau of Prints, disclosed no trace of fugitive sheets. There is one in St. Mark's Library in Venice, a very interesting copy of the female figure of Remellin, dated 1683, with numerous superimposed flaps. There is one known in Amsterdam, which I did not see, and a duplicate of this one is reported to be in the library at Copenhagen. There is one in the Surgeon-General's Library, the

[2] Studien zur Geschichte der Medizin, Leipzig, 1908, vol. iv.
[3] Geschichte der Gynäkologisch-Anatomischen Abbildung, Dresden, 1908.
[4] Wunder, Wundergeburt, Wundergestalt, Stuttgart, 1921.

Adam of the Vogtherr series, of about 1560, but unfortunately so imperfect that neither date nor printer can be determined. The Boston Medical Library, the Library of the College of Physicians in Philadelphia and the John Crerar Library, Chicago, have none. Dr. George L. Laporte, New York, has a copy of the Hela skeleton, 1493. Dr. J. G. de Lint, of Gorinchem, Holland, has several of these sheets, including a colored print of the viscera manikin, which first appeared in von Gersdorf's *Feldtbuch der Wundtartzney*,[5] and the very celebrated one representing the anatomy of man, in which the artist has used the features of Vesalius. He has a proof impression of Vogtherr's Eve, dated 1625, thirty years at least after any other known variant of the Vogtherr figure, and the Schonbornio skeleton, *circa* 1590.

I have been fortunate in securing ten definite early anatomical fugitive sheets, two of which are unique, and eleven others, Ryff's ten ten plates and the Hela-Gruninger skeleton, which may be placed in this same class. Two C. B. copperplates with a single superimposed flap were found pasted within the covers of an *editio princeps* of the *Fabrica*. Choulant[6] says that the copies of these plates which he had seen were cut to the border so that it could not be determined whether there was letter-press or additional figures upon the margin. My copies show no additional figures, but there is a column of letter-press at each side and a title above. The letter-press is in Flemish. Soon after making this discovery, I was offered through a dealer in Germany the squatting figure broadside. This is supposed to be unique as a fugitive sheet, although the same figure exactly appeared in smaller size in "Eyn Gert Artzney die hoc noch steet," 1525. Sometime later I found an unknown Italian fugitive sheet, dated 1611, laid in between the leaves of an elephant folio of Cowper's anatomy. I have the Wechtlin skeleton and viscera manikin, folded to fit, and inserted as illustrations in von Gersdorf's *Feldtbuch*. These figures are in perfect condition. Quite recently I was able to add four of these sheets of a rather late date: the

[5] Strasburg, 1517.

[6] Op. cit.

Vogtherr Eve, dated 1589; a remarkable Vogtherr Adam, being one of the two of these sheets which have survived, dated 1594; the Schonbornio skeleton and the Vesalius plate. The latter two, while less rare than the others, are nevertheless interesting. I have, of course, the Gruniger-Hela skeleton in my "Hortus Sanitatis," 1497. In addition, I was lucky enough to find neglected in a book shop in a western city a volume marked "Ryff, 1541." A careful study of this showed it to be made up of ten sheets printed on one side only and folded in the center to form pages, so that, without realizing it at the time of purchase, I had a complete set of Ryff's fugitive sheets which he published in 1541, and about which Vesalius voiced such a protest in his letter to Oporinus.

From a careful study of the descriptions given by Choulant, Sudhoff and Hollander, and a rather close inspection of the sheets, it would appear that these early anatomical fugitive sheets may be arranged in five classes as follows: (1) The skeleton sheets; (2) the Tabulae of Vesalius and its imitations; (3) the Adam and Eve plates; (4) the female figure alone; and (5) the male figure alone.

Sudhoff,[7] in his *Studien zur veschichte der Medizin*, has given a very satisfactory analysis of the evolution of the Hela skeleton. This and its subsequent modification, the Hela-Gruninger figure, he traces back to a drawing in a French manuscript in the Bibliothèque Nationale which was written in 1454. There can be no question after studying his figures and text that Sudhoff's genesis of the skeleton figure is quite correct. He cites the parallel development of the same idea in the cut of the skeleton in *Kalendrier des Bergiers*.

De Voynich and Garrison[9] have studied the use of the skeleton figures in the wood-cut borders of the early French books. The same evolution may be traced later in the figures of the "Dance of Death," which were so popular throughout the sixteenth century

[7] Studien zur beschichte der Medizin, Leipzig, 1907–1912.
[8] Geneva, 1495.
[9] Figurations of Skeletal and Visceral Anatomy in the Book of Hourz, Ann. Med. Hist., 1917, i, 225.

and of which Holbein was, of course, the master. In the skeletons of the "Dance of Death," it is not uncommon to see portions of the bony framework covered with shreds and tatters of desiccated epidermis, particularly suggestive of the effect of wind and weather upon the dead body. This might perhaps offer an explanation as to the source of anatomical material which was used as a model in drawing the Hela skeleton and its prototypes. In spite of anatomical inaccuracies of the skeletons seen in the famous French borders and the later cuts of the "Dance of Death," these frequently give a better impression of the true form of the skeleton than the more anatomically correct Hela-Gruninger blocks.

The Hela skeleton is first recorded as a fugitive sheet printed in Nuremberg, 1493. There was probably an earlier printing than this issued in Paris, since in all the known editions the author is given as "Richardus Helian, Parisiis." The British Museum copy, dated 1501, agrees in so far as the anatomical detail is concerned with the Nuremburg edition. The scrolls, however, are much more elaborate and a few show the legends reversed. The pediment is changed and the elaborate description in the upper right- and left-hand corners is boxed, and the mark of Wolfgang Steckel shows in the upper left-hand box. The feet in the 1501 edition have only four metatarsal bones, showing that a wood-cutter and not an anatomist made the changes.

The Hela-Gruninger skeleton shows slight changes in anatomy and the legends are printed in lines and not on scrolls. It is found in at least two incunabula, Braunschweig, 1497, and the Latin "Hortus Sanitatis" of the same date. Gruninger is supposed to have owned this plate, since the cut is used in so many of his books printed about 1520.

A distinct advance in anatomical detail is shown in the Wechtlin skeleton, which is definitely dated 1517. The skull, the vertebræ and the long bones are much more accurate, but the sternum, ribs and pelvis show little improvement over the Hela figure.

The Wechtlin skeleton and its companion plate, the viscera manikin, while issued as fugitive sheets, were inserted to be

used as illustrations in von Gersdorf's *Feldtbuch*,[10] and so are much less rare than the Hela prints. It is generally believed that Wechtlin was responsible for the viscera manikin. There has been considerable discussion as to who the wood-cutter of the skeleton was, as it is technically inferior to the viscera manikin. Choulant assigns this former figure to Grien, but the keeper of the prints in the British Museum believes that Wechtlin cut both blocks.

The viscera manikin marked an advance, not only on account of the beauty of its line but also because it represents the first anatomical fugitive sheet with smaller accessory cuts showing fine anatomical details.

In 1538 appeared the famous "Tabulae" of Vesalius, which were in truth wall charts, or "Fliegende Blätter," and represented an attempt to simplify teaching. All but the three skeleton figures, which were drawn by Calcar, are the work of Vesalius' own hands at a time when he was still trying to reconcile his own observations with the traditional authority of Galen. The popularity of the "Tabulae" is attested by the host of imitations which appeared within the next year. Vesalius complained bitterly of these plagiarisms in a letter to Oporinus and mentions six towns where the imitations were printed, but not the name of the authors or printers. Choulant, who devoted much attention to this, succeeded in identifying four of the authors.

I have had an opportunity to study but one of these imitations, that ascribed to Ryff. According to Choulant, Vesalius referred to Ryff in this part of his famous letter: "The fellow at Strassburg played a scurvy trick on scholars in that he so shamefully cramped the engravings which could not have been too large for the best results to scholars, and issued them as his own, wretchedly reproduced, and reduced in size out of all reason, with the text of the Augsburg version." The identification has been accepted by subsequent authorities, especially Roth, and so Ryff has come down in medical history as the superplagiarist of all ages.

[10] Johan Schott, Strasburg, 1517.

There were at least five different editions of Ryff's work between 1541 and 1545, a popularity which was not equaled by any of the other imitations of the "Tabulae" nor in fact by the "Tabulae" itself. The "Tabulae" was not reprinted and there were but two issues of the "Epitome," one Latin, one German, both in 1543. Two of the five editions of Ryff were as definite fugitive sheets, as was the "Tabulae." One, *Ossium Humani Corporis Descriptio*,[11] is printed with Latin letter-press, and is undoubtedly the original. The other[12] is the same except for a change in two plates. Choulant, in his discussion of the question, cites still a different edition.[13] In this edition there may have been different plates, since Ryff changed not only the arrangement but the plates themselves in the different editions.

A careful comparison of the first edition of this work with the "Tabulae" will show that Ryff used ten double sheets, on all of which, except the first, there are two plates, making a total of nineteen as against the six plates of the "Tabulae," with nine figures in all. Ryff's plates on pages *a* and *b* are definitely enlargements and improvements of the Dryander brain figures. Roth[14] says: "Ryff gives the skeletons; the plate of the veins, plate of the arteries, both drawn into a male figure; finally from the first plate of the "Tabulae" the male and female genitalia, both again drawn into the male figure." It is not claimed by Roth that the two figures of the muscle men are Vesalian. These two plates are adaptations from Da Carpi. At first glance the three skeleton sheets seem to be reduced and reversed copies of the "Tabulae," but a careful inspection will show that even in these Ryff used discretion in copying, since the thorax agrees more accurately with the plate of Dryander, which is dated 1537, than with the skeleton of Vesalius. Vesalius depicts the sternum composed of seven distinct bones, while Ryff, adapting the

[11] Strasburg, 1541.

[12] Paris, 1545.

[13] Des akkerfurtreffichsten, hochsten und adelichsten gschoppfs aller creaturem —das ist des menschen-warhafftige beschreiburg oder anatomi—erstmals in Deutsche sprach verfasset, Strasburg, 1541.

[14] Andreas Vesalius, Berlin, 1892, p. 123.

Dryander plates, represents the sternum as a single bone with the costal attachments, making it more correct than any of the "Tabulae" illustrations.

Even if the fundamental idea of these four other plates is Vesalian, improvement in placing the plates correctly in a figure drawn to scale and the necessary modification of the fundamental figure to fit the natural lines of the figures represent an advance, the credit for which must be given to Ryff. Unprejudiced study of the "Tabulae" and the Ryff plates will show that far from being a mere copyist, Ryff was a true editor. He undoubtedly selected what in his judgment were the best representations of the anatomy of the time for this particular set of plates, and that he did not use more of the "Tabulae" was simply because, in his judgment, the Vesalian plates were not an advance on the then well-known authority, Dryander. In fact, if one may speak candidly of a hero, the "Tabulae" of Vesalius did not represent a great advance in anatomical knowledge, however much they may have facilitated anatomical teaching.

I have been able to find, in various libraries, fifteen works which bear upon the title-page the name of Walter H. Ryff. These embrace not only medicine and surgery, but mathematics and architecture. Such opportunity as I have had to examine some of these books confirms the opinion above stated, that far from being a plagiarist, Ryff was in reality one of the best editors of his time, at a time when Erasmus, Gessner and Fuchs were proud to be considered mere editors.

Choulant cites extensively the other direct imitations of the "Tabulae" and a careful comparison of these imitations with the "Tabulae" would be worth study. Unfortunately these imitations are so rare that the work can only be done where the sheets are available for comparison.

Little need be said of the imitations of the idea of the "Tabulae." Quite a number of sets of fugitive sheets representing visceral and muscular anatomy were printed, some as late as 1580, but all of them represented only pre-Vesalian conceptions.

In some respects the *Epitome* of Vesalius may be classed as

fugitive sheets. While not fitting strictly the characteristics of a fugitive sheet, yet in this book Vesalius accepted the idea of the superimposed flaps introduced in the numerous fugitive sheets printed about 1539. The directions given at the top of the unlettered folios of the *Epitome* definitely show that Vesalius intended to have the figures on these sheets cut out and superimposed on the Adam and Eve plates.

The earliest of the Adam and Eve fugitive sheets appeared between the time of the publication of the "Tabulae" and of the *Epitome*, and all of them have the complete symbolism of the Garden of Eden. That this symbolism was thoroughly understood can be seen by following the evolution of the Adam and Eve idea in Vesalius. In the *Epitome* the skull replaces the apple in Adam's hand, but in the Geminus coppers which followed, in 1545, the apple is found in its normal position and the skull and snake repose at the left foot of the figure. In both of these cuts the fig-leaf is lacking, but in the female figure the hand of Eve naturally assumes the position of modesty which has since been conventional, and the apple and fig-leaf, the skull and the snake fitfully recur in the reprints and imitations of Vesalius up until the time of Remmelin.

This symbolism is not without its importance in any attempt to analyze the function of these fugitive sheets. Vesalius' reaction to this symbolism is quite remarkable. In the *Epitome* there is a skeleton followed by a beautiful Adam and Eve plate, but in the *Fabrica* his overcorrection causes the Garden of Eden idea to be omitted, but the three wonderful skeleton figures are so charged with the subconscious symbolism of the philosophy of death that it was recognized eighty-three years later by the genius of that age, who assimilated and transformed the idealism of these three figures into the graveyard scene of Hamlet.

Throughout this series of sheets, in which the female figure is shown with the fig-leaf, when the superimposed flap is lifted the pregnant uterus with the cowering fetus in the classical "See no evil" position becomes the high light of the entire figure. The Adam and Eve cuts show purely pre-Vesalian anatomy and

remained true to this type until 1590. Printed first with a single superimposed flap, within a few years there are several superimposed flaps, and the attention to detail and the accuracy in cutting and pasting are surprising. In Remmelin the culmination is reached and some twelve or thirteen flaps may be found on a single figure.

It has been a question as to which was the first of the Adam and Eve sets. Choulant cites the Sabio, the Guldenmundt and the Vogtherr sets as being printed in 1539. It is extremely probable that there was an example printed earlier, since the three similar sets appeared in the same year, and such coincidence would argue that these were copies of an earlier print.

The Jobst de Negker Eve, cited by Weindler and carefully studied by Wickersheimer, is definitely dated 1538. While no companion plate representing the male figure is known, it is possible that his was an inspiration for the numerous editions of the next year. It is, of course, possible that an Adam of this set may turn up, or a lucky discovery may even disclose an earlier set of plates embodying the Adam and Eve idea.

As far as I can find, the Sabio sheets were issued only once. The Guldenmundt cuts were reproduced in copper by Cornelius Bosch (Bos) and probably printed first at Antwerp about 1540. At least one other issue of this particular set of coppers is known with the text in English and with several flaps. The Vogtherr set, which was printed in 1539, seems to have been the most popular of all of these sheets, since it went through many editions. And this list is certainly incomplete, as I have been able to identify at least three issues not mentioned by Choulant. Choulant gives his inclusive dates from 1539 to 1590, but there is a copy of the Adam plate dated 1594 and of the Eve plate as late as 1625.

The Adam and Eve plates reached their culmination with Remmelin, first published in 1613. The fugitive sheets after Remmelin show the female figure alone, a retrogression in the later history to a type of illustration almost identical with the early fugitive sheets inspired by the squatting figure of Ketam.

The squatting posture as shown in the famous Ketam illustration is the distinctive feature of the fugitive sheets showing the female figure alone. This froglike attitude is found in each one of the set of five drawings which Sudhoff has so carefully studied. He attempts to trace this set of anatomical illustrations to Alexandria. While, of course, no drawing of this epoch is preserved, Sudhoff's argument is convincing, and undoubtedly in this set of drawings we have a fair idea of the extent of anatomical knowledge at Alexandria. The most beautiful of the codex drawings of the female figure is that of the Leipzig manuscript number 1122 (1400), which has been used as an illustration both by Weindler and Choulant-Frank. In this particular miniature there is a slight deviation from the froglike posture, but the pregnant uterus with the cowering fetus is the prominent part of the picture. All the peculiarities of posture in the set of five are retained in the Ketam illustration (1493), which is one of the most beautiful of the famous Venetian wood-cuts, and such an advance over anything previously done that it is generally considered to be the first drawing made directly from a dissection. The squatting posture persisted in sheets showing the pregnant uterus, but there are at least two intermediate stages between the latest of the five-figure series and the 1493 Ketam.

Dr. de Lint has a manuscript fragment of the Ketam which he dates in the middle of the fifteenth century. In addition to text, there is shown the lower half of a figure in the typical posture, but with the details of the gravid uterus much clearer than in the Leipzig miniature.

The figure in the 1491 Ketam is an advance over this figure of de Lint's, but approaches neither the correctness of anatomical detail nor beauty of line of the figure of the 1493 and subsequent editions of Ketam. This same figure, coarsely copied by an indifferent wood-cutter and modified by use of parallel lines for shading, is probably the earliest of the fugitive sheets of this class. It may be dated at least ten years earlier than the first of the Adam and Eve sheets. The wood-cut is accompanied by a page of letter-press with the description of the various organs

in Latin. The fugitive sheets printed later did not always show the typical froglike posture, but the de Negker print, my Italian sheet, and even the copy of Remmelin in St. Mark's Library show attitudes which are nothing more than deviations from the squatting posture. The same fundamental idea can be seen in Stephanus' plates, and in fact persisted in anatomical book illustrations even as late as Cesaerius, whose plates evoked Oliver Wendell Holmes' famous caustic comment. The Eve of the Guldenmundt and Vogtherr series also represented figures in attitudes which are closely related to this same posture.

So many more of the Eve than of the Adam sheets have survived that it seems extremely probable that even during the period of the popularity of the Adam and Eve sheets the female figure was more frequently printed. The female figure alone followed closely in time sequence the skeleton figures and antedated the first of the Adam and Eve plates. The skeleton figures disappeared almost completely upon the publication of the "Tabulae," while the Adam and Eve sheets were printed in great numbers up to 1590 and even later. The female figure persisted for nearly another century.

It is a natural inference that curiosity was the force which led to the production of such a large number of these sheets, and this curiosity was concerned particularly with the details of the female anatomy. Even of the popular Vogtherr plates many more of the Eve sheets have survived, and later only sheets representing the female figure alone were printed, at least so far as any traces are known, in the fugitive sheets after 1590. We may likewise notice evidence of progression. At first the only details worked out carefully were the reproductive organs, and in the female figures the uterus is represented as enlarged, with a window showing the fetus. This tendency exists through the entire series of the Guldenmundt-Vogtherr sheets, but by the time of Remmelin it was necessary to have three plates—the male, the female and the pregnant torso. In the sheets following Remmelin the female figure alone is represented with no appearance of pregnancy, a very normal correction of primitive curiosity.

The only sheets representing the male figure alone are those printed by Schonbornio at Viiteberg about 1590. This was at a period when the Vogtherr Adam and Eve sets were still in great demand, but it appears that Schonbornio was influenced by quite a different idea. His male figure is perhaps a modification of the Vogtherr plate, but the posture of the seated figure is quite different and the wood-cutter has attempted to replace the bearded head of the older cuts with the features of Vesalius. Not satisfied with the first result, the artist changed the famous hair-line and the beard in each new issue of the wood-cut. There are at least two and possibly three variations of this plate which differ only in the shape of the beard and hair-line, and it would appear that the wood-cutter had tried to reproduce from memory the features of the great anatomist. Schonbornio also edited a plate of the skeleton which was a modification of the anterior view of the skeleton of the *Fabrica*, so it may be assumed that Schonbornio's admiration for Vesalius was the reason of his activity in printing these fugitive sheets. It has been suggested, however, that the features of Vesalius were placed upon these sheets as a guarantee of the correctness of the anatomy represented, but the visceral anatomy shown on the three superimposed flaps has many features definitely pre-Vesalian and the changes in the skeleton drawing are not a great improvement.

For the most part, fugitive sheets were printed upon a good quality of soft paper, rather thicker than that used in the making of books. The size generally approximated about 30 by 40 cm. The main figure is usually surrounded by descriptive text, though occasionally an extra sheet is added for letter-press. Decorative borders are not uncommon. Some of these borders are attractive in a general way, and occasionally the border approximates in beauty the best traditions of the printing art. The use of smaller cuts surrounding the main figure was introduced in the Wechtlin manikin and the use of these cuts prevailed in most of the sheets, although with variations in number and kind even in similar sheets. The newborn babe is found sitting beside Eve in the Vogtherr series and is very attractive. It is reminiscent of the

Della Robia babies and gives the same touch to this picture as does the famous woman with the vase to the Zodiac figure of Ketam. Variations in spelling are an indication that they were printed before there was fixation of language, rather than evidence of bad spelling or copying. The general outline of the principal figure persisted fairly true to type throughout the whole history of an individual class. Innovations and improvements were concerned more with the details of the superimposed flaps than with any other part of the sheet. The single flap is found first in several fugitive sheets issued in 1539; at a somewhat later period we find two or more flaps, and during a still later period four or more flaps. These flaps generally show great care in printing, cutting and pasting. The anatomy itself, however, does present some evidence of advance, particularly over the crude wood-cut of the graphic anatomical incunabula. In the prints of the later decades of the sixteenth century there is a fairly accurate outline of the thoracic and abdominal viscera, but even in the latest the horizontal diaphragm is still in evidence.

In the sheets of the earlier period the printing is usually in black, but a few of the earlier sheets may be found colored by hand. Comparatively early some attempt at color printing was made, and many of the later sheets represent fairly successful efforts at printing in colors.

Most of the classes show at least one example printed in Latin, but the other issues of the same block have the text in the vernacular. It is probable that the first edition was in Latin and that the text was translated into the vernacular in subsequent editions. This is perhaps a further indication that sets antedating the Vogtherr and Guldenmundt issues of 1539 have been lost.

There are so few sheets extant that it is extremely difficult to identify the city of origin. Even the Hela-Nuremberg print was probably a copy of an original from Paris, since Hela is spoken of as "Helian Parisiis." There is no doubt that later Paris took a lively part in the production of these prints. The printers of Venice also are represented in the check list, but the majority of

the sheets are from the presses of the German cities actively engaged at the time in cheap printing. Few, if any, of the famous presses are represented in the list, nor do we have sheets from the printers of the University cities, as would have been the case were these sheets used for purposes of anatomical teaching.

The Wechtlin sheets are much more common than the other broadsides, since they were used for inserts in the *Feldtbuch* and consequently have been preserved in the book itself; but even these were printed in different sizes for different editions by Gruninger, and the variant in the British Museum shows that they were modified and issued by different printers. At least three variants are known of the skeleton and two of the viscera manikin.

The Hela skeleton has at least one variant, that in the British Museum, in which the distinctive feature is the presence of only four toes. The Schonbornio manikin, to which was added the features of Vesalius, shows two and perhaps three attempts to reproduce the likeness of Vesalius, and a careful study of these different issues will show typographical corrections and changes in fonts sufficient to indicate resetting of the letter-press as well as alterations in the wood-cut. Dr. de Lint attempts to argue from the title of the Schonbornio skeleton, beginning "Seu compages ossium humani corporis," that it was one of a series probably of three plates, but in his copy a black border blots out the first work of the title *ΣΚΕΛΕΤΟΝ*, which seems to explain the use of the word "or."

In fact, in all the other fugitive sheets studied no two are precisely alike, and each new sheet which happens to come into the market is described as unique. This would argue that many editions may have been printed of which no copy survives, since of such editions as are known only one copy has survived.

These sheets were issued at a time when there was no copyright protection, and comparison shows that the original plates were copied sometimes exactly, sometimes carelessly, sometimes fantastically, and published by different printers, and that such changes went on successively until the original figure was altered

beyond recognition, and that the accessories, the form of the letter-press, the language, the decorations and even the figures themselves were altered to suit the whim of the wood-cutter or printer.

In the few books in which we find reference to these fugitive sheets their use has been a matter of but casual speculation. Choulant says that they were used for display purposes in barber shops, bath houses and apothecary shops. Considering the very large number published, this would have been a very narrow outlet. Dr. de Lint has recently investigated the question and his explanation is worthy of consideration. He believes that they were printed for the use of students, practically as are our quiz compends of today; that books were too expensive for many of the students and some cheaper substitute had to be found, and this substitute was the fugitive sheet. As a further argument, he states that in the latter part of the sixteenth century there was a marked decrease in the price of books on anatomy, and that coincident with this decrease the fugitive sheet went out of existence.

While undoubtedly these sheets were hung in various places, as mentioned by Choulant, and while they may have been printed for the use of the students, there must have been a much wider distribution of them than could possibly be represented by the ideas of either Choulant or de Lint. Examples are very scarce at the present time, and yet the very number of different wood-cutters, presses and printers represented in the sheets that we do know argues an extensive distribution in the sixteenth century. That they were not, primarily, for medical students is shown by the large proportion which are printed in the vernacular at a period when the medical student prided himself upon his Latin, and when medical instruction was wholly in Latin, save the departure that had been made by Paracelsus. It was thoroughly understood at that time that an education was necessarily an expensive luxury, and it is therefore hard to believe that medical students would buy and study, fifty years after the wonderful books of Vesalius and his successors were available, anatomical

compends printed in the vernacular and typifying Galenic anatomy.

Viewed from all angles it would appear that these sheets were printed not for students but for the common people, and printed in large quantities because they were anxiously sought for by the people in general as giving a long-sought answer to a question based upon a primitive psychological desire. In support of this interpretation one might cite the large number of these sheets printed in a common language. There are fugitive sheets printed in German, French, English and Italian, which would show an almost universal demand for such information as could be furnised by this means. If, as Jung states, our own psychological development represents the development of civilization itself, we can easily assign to the Renaissance the period of adolescence. And one of the great queries of adolescence of the race or of the individual is, "Where do I come from?" The titles and legends of these early fugitive sheets seem further to confirm this position. The title, "Abconterfeyung eins Mann's Leib, wie er inwendig gestalt ist," etc. (portrayal of a man's belly, how he is formed internally; and of a woman's belly, how she is formed internally), is found in the entire Guldenmundt and Vogtherr series, and the legend, *Nosce te ipsium* (Know thyself) is frequently found on the French prints. Further, as we study the popular Adam and Eve and the squatting-figure sheets, beginning with Ketam and going through the Guldenmundt series up to and including the sheet at St. Mark's, we have before us a picture in which the greatest importance and stress are laid upon the female organs of generation, and in which the pregnant uterus with the fetus in the classic position is the central part of the arrangement. This was not, I think, a pornographic appeal, but, as I have suggested, was to meet an interest in a question as old as the world and fundamental in the religious and artistic development of the time.

To me this seems to justify the conclusion that the people in general were so eager to get such information that they demanded these fugitive sheets, and the demand was met by edition after

edition and print after print, from the presses the country over, of pictures which would seem, at least in a physical sense, to give an answer to this question.

Check List of Recorded Fugitive Sheets

The following check list is based primarily on Choulant's original description of these plates. The additions made by Frank have been incorporated and also the sheets described in accessible literature. A description of the fugitive sheets (several not previously recorded) found in various libraries and private collections, as well as those of my own collection, have been included. The list is rearranged to fit the classification considered above. After each formal title is listed the authority which has been cited in this article. Where no asterisk is inserted the sheet has been described only; a single asterisk following the name indicates reproduction by authority quoted. Those seen and studied by the author are marked by two asterisks, and those in the author's collection are marked by three asterisks.

CLASS ONE.—SKELETON SHEETS

TYPE I.—SINGLE SHEET: THE HELA SKELETON

Anathomia ossium corporis humani. Nurenberg, 1493. 53 cm. high. Letter-press in Latin. (Choulant,* Sudhoff,* Hollander.*)

This is the earliest known fugitive sheet, although it is quite probable that a similar sheet antedating this one was printed in Paris, since the sheet is attributed to Richard Hela of Paris. From many standpoints it is the most important of all the sheets, and its part in the development of anatomy and anatomical illustration has been carefully considered by Choulant, Sudhoff and Hollander.

Variant A: Same title. 35 x 25 cm. The bony parts of the skeleton have been tinted a light ochre. It is dated Nurenberg, 1493. (Dr. George L. Laporte, New York.**)

Variant B: *Anathomia ossium totius humani corporis.* Leipzig, 1501. 33 x 22 cm. Letter-press in Latin. (British Museum.**)

Underneath the title, which is printed above a black border, is a subtitle, "Anathomia ossium." There are some slight variants in the figure,

especially noticeable in the feet, which have only four tarsal and metatarsal bones. The letter-press in the upper corners has a different arrangement and the block in the upper right-hand corner has the Wolfgang Stoeckel printer's mark. In the upper left-hand corner there is a scroll instead of the square block, in which is the date 1501. The scrolls are much more elaborate, and in several the Latin name is inverted. The pediment is larger and more distinctly outlined.

VARIANT C: *Anathomia ossium corporis humani.* Strassburg, 1497. (Choulant.*)

The so-called Gruninger block, modified from the original Hela, reduced in size. Has the names of the bones printed in simple type lines, instead of the scrolls of the original. This figure was perhaps a fugitive sheet, but it is frequently used as a book illustration. The first that I have been able to identify is in the "Hortus Sanitatis," 1497, and Braunschweig, same date. This block appeared in many of the Gruninger books published from 1520 to 1530, and is supposed to have belonged to him, as he used it indiscriminately.

TYPE II.—TWO SHEETS: (*a*) THE WECHTLIN SKELETON; (*b*) THE VISCERA MANIKIN

(*a*) *Ein contrafacter Todt mit ein beimen fugen und glyderen und gewerbem, vsz beuelh loblicher gedachtnusc hertzog Albrechts bischoff zu Strassburg, durch meister Nichlaus bildhawer zu Zabre worlich in stein abgehawen, und noch anszoig rechter gewisszer anatomy mit sein latinischen namen verificiert.*

Inserted in *Feldtbuch der Wundtartzney,* by Hans Gersdorff. Strasburg, Johan Schott, 1519. 36 x 27.5 cm. Letter-press in German; anatomical names in Latin. Printer's mark of Johan Schott.***

Front view of the skeleton, with head slightly turned to right, arms hanging down. Latin names of bones cut on plate. In the upper left-hand corner the date 1517. Below the date is printed twenty-four verses of moral reflections upon death.

This was designed as a fugitive sheet and not intended for a book illustration, but it was folded in the center and bound in Gersdorf, as given above. A wood-cut struck from the same block is also folded and bound in Phryesen's *Spiegel der Artzney.* Strasburg, Joh. Gruninger, 1518.

(*b*) Viscera Manikin. *Anatomia corpori humani,* 1517. Above the plate is printed, "*Ein contrafact anatomy der inneren glyderen des menschen durch den hochglerten physicum und medicine doctorem Wendelinum hoch von Brackenaw zu Strasburg, declariert und eygentlich in beysein viler Scherer un Wundartzt grundlitch durchsucht.* Johan Schott, Strasburg,

1517. 36 x 21.5 cm. Letter-press in German; anatomical names in German. Below the plate, forty-six verses printed in three columns.***

This plate is also folded and inserted as a book illustration in Gersdorf's *Feldtbuch der Wundartzney*, Strasburg, 1519.

The plate was drawn and engraved by Hans Wechtlin (Veuchtlin), of Basel, called the master with the crossed pilgrim's staves, on account of his monogram. The drawing excels the Skeleton (*a*), which was evidently done by a less-skilful hand, although attributed by the keeper of the prints in the British Museum to the same artist. This peculiar dissection with the five-point liver and a perfectly horizontal diaphragm may be followed through most of the fugitive sheets up to 1600. This is also the first introduction of accessory figures, which persisted throughout the entire history of fugitive sheets.

Variant A of (*a*): Same title.—This sheet is undated and does not have the printer's mark. The typographical arrangement of the title and verses is slightly different; a few slight changes in shading. It is 33.5 x 25.5 cm. Letter-press in German; anatomical names in Latin. (British Museum.**)

According to Choulant, this same wood-cut, without title or verses, appeared in Phrysen's *Spiegel der Artzney*. Strasburg, Gruninger, 1518. Dr. Harvey Cushing showed me the same cut in Albucasis, Johan Schott, 1532.

Variant A of (*b*): Viscera Manikin.—Title cut off, date omitted, a few changes in spelling of legends; the poetry in three columns, and no line between the figure and verses. (De Lint.**)

TYPE III.—SINGLE SHEET: SKELETON

Sev. compages ossium humani corporis. Parte anteriore expressa. 30 x 39 cm. N. D. Black line border. Letter-press in Latin. (De Lint.**)

This plate is definitely assigned to Shonborni, and therefore must be dated about 1590. Front view of a skeleton with the right arm resting on the grave-digger's spade and the left hand supporting the skull with the base presenting. The sternum shows five divisions. Surrounding the main figure are seven smaller figures: at the right, lateral view of the skull; to the left, front view of the sacrum. Letters drawn on the bones refer to the accompanying text, which surrounds the wood-cut.

This skeleton is a direct adaptation of the Vesalius skeleton in the *Fabrica*. The skull is tilted forward and the left arm elevated, which is not found in the original. The general drawing is not as good as the Vesalius skeleton.

ΣΚΕΛΕΤΟΝ VARIANT A.—*Sev compages ossium humani corporis. Parte anteriore expressa.****

Exactly the same figure; practically the same letter-press. A few typographical errors have been corrected. Certain of the subtitles are printed in upper case in Roman letters instead of upper case italics.

TYPE IV.—TWO SHEETS

Osteotome i. ossium humani divisio ex Galeno praecipue. Paris: Christianum Wechelum, 1536. Folio. Letter-press in Latin. (Choulant.)

Front and back view of erect skeleton in wood-cut; drawing better and more exact as to anatomy than Da Carpi, Dryander or Ryff. The wood-cut is beautiful and distinct.

These two sheets can also be found in the Greek edition of Galen, Basel, 1538. Folio v.

TYPE V.—SINGLE SHEET: SKELETON

By il Simoleo. 41½ x 30. (Choulant.)

Copperplate engraving of the skeleton by Giovanni Battista Franco (called il Simoleo), representing a profile view of a human skeleton down to the knees, looking to the left and extending half-way into the border, showing at the top a skull and on the right side the bones of the extremities.

Franco was born in 1498 and died in 1561 or 1580.

CLASS TWO.—THE TABULAE AND IMITATIONS

TYPE I.—SIX PLATES

Des Andreas Vesalius sechs anatomifche Tafeln vom Jahre, Imprimebat B (ernardinus) Vitalis, Venetus, sumptibus Joannis Stephani Calcarensis. 1538. Large folio. Letter-press in Latin. (St. Mark's Library, Venice.**)

These are probably the most famous of all fugitive sheets. As is well known, only two sets exist—one in St. Mark's Library, Venice, and the other owned by Sir Sterling Maxwell, who made an exact reprint and published thirty copies, which he so generously distributed that most of the large libraries have a copy.

More recently Holl-Sudhoff[1] reproduced these plates in a large edition.

[1] Leipzig. 1920.

Unfortunately, difficulties in printing required that they be reproduced in three-fourths size of the original.

After Vesalius issued these plates, in 1538, many imitations appeared, some merely copying the idea and others being direct plagiarisms of the plates.

TYPE II

Jobst de Necker: *Ein gar kunstlich allem Leib- und Wundarrzten nutzliches werk 6 figuren mit inhalt aller blut-Schlag und Flechsadern sant den Gebeinen des ganzen Leibes, Augsburg,* 1539, 6 leaves; folio. Letter-press in German. (Choulant.)

The work of the Augsburg wood-engraver, Jobst, or Jobst de Necker (Negker). This sheet, dealing with the veins, arteries and nerves, is by all means the truest and best copy of Vesalius' first six plates. However, in the first six plates of Vesalius there was none of the nerves, and inasmuch as the title of the Necker sheet has the word "Flechsadern," it is probable that the Macrolios sheet was added.

TYPE III.—TWELVE SHEETS

Ausslegunk und bedchreybung der Anatomia. Gedruckt, zu Nurenberg, durch Hans Guldenmundt, 1539. 4to. Letter-press in German. (Choulant.)

Twelve sheets, with crude wood-cut engraving between the text, representing the organs of the thoracic and abdominal cavities, conveying popular instruction in anatomy and remedy for disease. There are no entire figures. The wood-blocks were made by Hans Weygel. It was republished at Ulm, 1541.

Guldenmundt was a wood-engraver and printer in Nurenberg between 1520 and 1546.

TYPE IV.—SIX SHEETS

Ein gar kunstliche, aller Leyb und Wundartzen, auch andrer kunsten Liebhavern, hochnutzlichs Werk in sechs Figuren gebracht, mit kinhalt aller blutchlag und Flechsadern, samp den gebaynen des guntzen Leybs. Cologne, N. D. Folio. Six leaves, printed on one side only. Letter-press in German and Latin. (Choulant.)

Below the preface is a rather large engraving showing the liver and spleen; also two smaller engravings showing the genitals of the two sexes.

The second sheet contains a large wood-cut of the liver and the vena

cavæ, with their roots. The third sheet shows the heart, kidneys, aorta and its branches. The other three sheets show the skeleton viewed from front, back and sides.

The engravings are effective, those of the skeletons being particularly good; but all of the anatomy is pre-Vesalian, showing the liver with five or six lobes and the heart situated directly in the center, after the manner of Galen; the uterus is now shown without horns. The skeletons are colored gray and the background illuminated. Necker's name is not mentioned, but this may be a copy of his work.

TYPE V

Aegidius Macrolios: *Cerebrum animalis facultatis fons et principium sensum voluntarium per nervos communicans ab se et dorsali medulla enatos universo corporso.* Cologne, 1539. Letter-press in Latin. (Choulant.)

This is a sheet printed on one side, representing the brain, natural size; the upper part cut horizontally open so that two lateral ventricles become visible; beneath the brain are the cranial nerves, also tongue and a piece of the palate. Drawn in a bold and free manner; nerves unnaturally enlarged; some of the words and letters cut in wood, other printed.

This sheet must have been published in 1539, and the publisher probably had only Vesalius' hand drawings or a copy in his possession. This plate is not among the first six Vesalian plates, which represented the system of the veins and arteries.

The name Macrolios was not known to Vesalius. The latter seems not to have seen this very rare sheet, but only a copy of it. But that the drawing was Vesalian is shown by the cut (page 319) of the 1543 *Fabrica* and that of the 1555 edition (page 512).

TYPE VI.—TEN SHEETS

Omnium humani corporis partium descriptio. M. Gualtheri H. Ryff, Argentini in Medici, Ex-officina Libraria. Bathaffis Piftoris, 1541. 29 x 19 cm. Letter-press in Latin.***

This consists of a set of ten double-folio sheets, labeled A–E and i-e. The left half of the first folio contains title, address to reader and place and date of printing. At the top of each subsequent folio is an anatomical description with letters which refer to the initials upon the plate itself. The right half of A shows the male viscera with lines and letters. The female figure is similarly shown on the left half of B. The next two cuts are the front and back view of the muscle man, quite evidently adopted from Da Carpi but without letters. On the right half of Folio

C is shown the venous circulation, with numerous lines and initials but without adequate accompanying text. The arterial system is shown in a similar way on the left of Folio D. The anterior view of the skeleton appears on the right of Folio D, and the side and back view on Folio E. In these three plates the figures are lettered but without accompanying text.

Folios A–E show the brain figures of Dryander, enlarged and reversed from the original, and there is added, as a decoration, cuts of various anatomical instruments. All of these figures are very much superior to the original Dryander figures.

Variant A: Same title. Paris, 1543. Letter-press in French. Plates same size; letter-press on both sides of each sheet and a few pages in addition. Two plates of the circulation which are not in the original. (British Museum.**)

Variant B: Same title. Paris, 1545. 44 pp. Letter-press in Latin. Same plates as in the original but in different sequence. The text extended and a portion printed on reverse of plates. (Dr. Harvey Cushing.**)

Variant C: Same title. Paris, 1545. Ten double sheets, same size. Letter-press in French. The muscle men are from entirely different wood-cuts; the other plates are the same. (College of Physicians, Philadelphia.**)

Variant D: *Des Allerfurtrefflochsten, hochsten und adelichsten gschoppffs aller Creaturen- das ist, des menschenwarfftige beschreibung oder Anatomi-erstmals inn Teutsche sprach verfasset.* Durch M. Gualtherum Hermeneium Ryff, Argentinum Medicum, 1541. Colophon: Strassburg, bey Bathassar Beck. Folio, 6–73 11. Gothic type. (Choulant.)

This contains, including repetitions, twenty-five anatomic plates in wood-cut; ten taken from Dryander, redrawn and enlarged; the other fifteen being full-page cuts of the skeleton, front, back and sides, drawn with a rather inferior understanding of anatomy; two muscle manikins, two reproductions of the bloodvessels of the whole body; one plate representing anatomic instruments, and fifteen smaller figures taken from the Rose Garden, showing the position of the fetus, and finally a schematic reproduction of the eye.

TYPE VII.—EIGHT COPPERPLATES

Skeletons and muscle manikins, with the signature, Ph. Galle, *fecit ex exudit.* Probably these plates were used later in *Instructions et foundamens de bein pourtraire pour les pentres.* Antwerp, 1589. (Choulant.)

Galle was born in 1537 and died in 1612.

TYPE VIII.—FOURTEEN COPPERPLATES

Muscle manikins, skeletons, etc. Guilio Bonasome. 4to. (Choulant.)

These plates contain artistic anatomy and were designed, drawn and engraved by Guilio Bonasome, who lived in Bologna from 1520 to 1580. Each plate represents the muscle manikin, the fourteenth being a skeleton with the left side still covered with flesh. The representation of the muscles, on the whole, is good, but the skeleton is very badly drawn. The positions are varying but always picturesque and lifelike. Some of the figures are holding in their hands a staff or rope, others their flayed skin. The background of all the plates is crosshatched. Some have the monogram J. V. B.

CLASS THREE.—ADAM AND EVE PLATES

TYPE I.—TWO SHEETS

Nicolai de Sabio viscerum viva delineatio. Venice, 1539. Folio.

Male and female, with the abdominal viscera drawn on separate, movable layers. The drawing is crude, showing the liver with many lobes. (Choulant.)

TYPE II.—TWO SHEETS

Anatomia oder abconterfectung eines Mans leib, wie er inwending gestalt ist. Gedruckt zu Nurenberg, durch Hans Guldenmundt. Folio (1539). Letter-press in German. (Weindler*—Choulant.)

Adam and Eve figures: man with twig in his right hand and apple in left; woman has a flower in her left hand. Both nude and seated. Pre-Vesalian anatomy. The anterior wall of the trunk can be opened and turned upward. In the female figure we have the uterus enlarged and opened, with the cowering fetus with hand before the eyes. On the female figure are the names of the different organs; on the male figure, the letters only. Both figures show crosshatching. The drawing was probably done by the wood-engraver, Peter Flotner, of Nurenberg, who died in 1546. Weindler gives the date as 1539. The sheet in his book is colored and has a small figure at the side, but it is defective, as the fig-leaf has been torn off. The sheet cited by Choulant was not colored.

TYPE III.—TWO SHEETS

Title not cited. Folio. Copper with monogram C. B. (1539). A direct copy of the Guldenmundt sheets in copper, probably by Cornelius

Bosch (Bos), a copperplate engraver of the Nederlands, who moved from Antwerp to Rome in 1540. These sheets were probably engraved before his departure.

Choulant says: "I am unable to see from the copy before me whether text and illustration surround the main figure, the edges of my copy being close to the margin."

Both sheets are colored. (Choulant.)

Variant A (two sheets): *Anathomia oft deylinghe eens Mans lief, also si beinnen ghestelt is.* Second sheet the same, except "*Voijfs*" (instead of *Mans*). Folio. Monogram C. B. in copper. N. D. Letter-press in Flemish. 40 x 39.5 cm.***

The male figure has the monogram C. B. at the lower right-hand corner. At the end of the text surrounding the female figure are the initials I. O. B.

This copy is uncolored. No accessory figures surrounding the main figure. On the male figure the various organs are lettered; these letters refer to the text, which is printed in columns on either side and below the main figure. In the female figure the names are engraved upon the organs and the description is printed under a similar name in columns at the side only. There is of the female figure two sheets, one being merely the figure alone, and another a large flap which lifts up showing the organs printed directly upon the foundation sheet. The male figure is similar, save that there is one large superimposed flap.

Variant B (two sheets): *The Anathomye of the inwarde Parts of Man lyvyely set fourthe and dylegently*, and "The Woman." Folio. Letter-press in English. Black letter. Copper (1550).

These sheets are possibly from the same C. B. plate, but unfortunately they are defective just where the monogram shows in the original. There is no letter-press, but a very interesting border occupies the position of the title in the original plates. There are several flaps on each figure, which indicate that the date is probably about 1560. (Wellcome Historical Medical Museum.**)

TYPE IV.—TWO SHEETS

Anathomia, oder anconterfectung eines Mans leyb, wie er innwendig gestaltet ist . . . eines Weybs leyb, wie er innwendig gestaltet ist. Getruckt zu Strassburg durch Heinrichen Vogtherren, 1539. Folio. Letter-press in German. (Weindler*—Choulant.)

Man and woman, nude, sitting posture; some kind of garment thrown across the hips. Apple and flower lacking. Front wall of trunk can be folded upward. Different pictures of the various organs can be lifted

up separately. Names of organs printed either in Latin or German. Crouching fetus in the uterus, with hands at side of the head but not covering the eyes. The drawing is more crude and the anatomy more obsolete than that of the Guldenmundt sheets. There are some illustrations as on the Guldenmundt, but from a different wood-engraver.

Two wood-engravers and art dealers in Strassburg had the names Heinrich Vogtherr. The older one was born in 1490 and the younger in 1513. These sheets are probably by the older man.

VARIANT A (two sheets): *Anatomia interiorum partium humani corporis ac earundem situs, figura, numerous, positio, haud, iniucunda cognitu-Anatomiae perutilis interiorum mulierbris partium cognitio ac earundem . . . cognitio.* Argentorati, a pud Jacumbum Jacundum, 1551–1552. Folio. Letter-press in Latin. (Choulant.)

Representation of man and woman. Latin text on back; smaller representation of the organs within. Principal figure identical with those published in 1539 by Vogtherr in Strasburg. Plate of male figure, 1551; female figure, 1552. Both plates illuminated.

VARIANT B (two sheets): *Anathomia oder abconterfectung eines Mans leib, wie er inwendig gestaltet ist . . . eines weibs leib, wie er inwendig gestaltet ist.* Getruckt zu Nurenberg, durch Hans Weygel, Formschneyder, 1556. Folio. Letter-press in German. (Choulant.)

Exact copies of the figures of Vogtherr, but different and inferior wood-cuts. Internal anatomy also demonstrated in exactly the same way. The smaller wood-cuts between the text that surrounds the main figure are the same drawings but reëngraved. Several of the wood-blocks have been used before in Guldenmundt's book, *Ausslegung und beschreybung der Anatomi*, Nurenberg, 1539. Both sheets are illuminated. Hans Weygel, of Augsburg, was a wood-engraver and art dealer of Nurenberg. He died in 1590.

VARIANT C (two sheets): *Anathomia oder abconterfectung eins Mans Lieb wie er inwendig gestaltet ist . . . eines weibs lieb, wie er inwendig gestaltet ist.* Gedruckt zu Nurenberg, durch Matthes Rauch Brieffmaler, 1584. Folio. Letter-press in German. (Choulant.)

Wood-cuts from the Weygel plate. Arrangement of the sheets the same. Female figure illuminated.

VARIANT D (two sheets): *Anathomia oder abconterfectung eines Mans Leib, wei er inwendig gestaltet ist . . . eines weibs leib, wei er inwendig gestaltet ist.* Gedruckt zu Frankfurt an Mayne, bey Conrad Corthoys. s. a. Folio. Letter-press in German. (Choulant.)

This drawing repeats the Vogtherr figure. The wood-cut of the main figure and smaller wood-cuts are new. All around the sheet runs a decorated border. Within the border is the explanation, with smaller

wood-cuts on both sides, of the male figure. The male figure is illuminated.

VARIANT E (two sheets): *Anathomia, oder abconterfectung eines Manns Leib, wie er inwendig gestaltet ist . . . eines Weibs Leib, wie er inwendig gestaltet ist.* Gedruckt zu Nurenberg, bey Georg. Lang (Formschneider), 1588. Folio. Letter-press in German. (Choulant.)

This is also the Vogtherr figure. Plate made by Hans Weygel. The male figure is illuminated. Pre-Vesalian anatomy.

Georg. Lang (Formschneider), wood-cutter and illustrator, is supposed to have lived in Nurenberg and died about 1620.

VARIANT F (single sheet): *Tabula Foeminae Membra Demonstrans.* Vitberg, Simonis Groneberg, 1589. Folio. 40 x 29.5 cm. Letter-press in Latin.

The figure itself, which occupies the upper left-hand corner, is 32 x 18 cm.*** The arrangement of the accessory figures is much the same; those below the figure are practically identical. The newborn babe still sits on the bench to the right of the main figure. Above, however, there are two accessory figures, instead of one as in the original Vogtherr plate. On the left, a cut of the stomach replaces the original figure of the female genitalia, and to the right is a crude cut of the posterior wall of the stomach in contact with the spleen. The figure is illuminated. This is probably the Eve of the Vogtherr Adam and Eve set.

VARIANT G (single sheet): *Anathomia oder abconterfectung eines Manns Leib wie er inwendig gestalt ist.* Nurenberg, Georg. Lang, 1594. 54.5 x 29.5 cm. Letter-press in German. Folio.***

Made up of two sheets pasted together, printed in part in colors, the balance illuminated. The main figure occupies the lower center of the sheet; several superimposed flaps, of which only two remain. The main figure is surrounded by eight labeled but not numbered smaller figures with the Latin name. The text is in three columns. A very interesting feature of this particular sheet is that the head and abdominal flap are printed from the same sheet and pasted over the fundamental sheet, upon which is printed the outline of the body.

The male figure of the Adam and Eve set is now very rare. There is one in the Surgeon-General's Library which, unfortunately, is so defective that it shows neither date nor place of printing.

VARIANT H (single sheet): Same title. Folio. The female figure alone. Viiteberg, Johannis Germans, 1625. 43 x 32 cm. Letter-press in Latin; text in columns to right and left. (De Lint.**)

This sheet is of extraordinary interest, inasmuch as it extends the history of the Vogtherr series thirty-five years after the latest example

cited by Choulant. This particular sheet seems to be a galley proof; another proof with letter-press alone has been drawn on the reverse of the sheet.

TYPE V.—SINGLE SHEET

Anatomie tres-vtile, pour coggnoistre les partes interieres de l'homme et de la femme. Composes par Maistre Andre' Vesali, ausc ample declaratione des veynes principales et manier de beine Signer. Paris, apud Jean de Bourmont, 1585. Folio. Letter-press in French. (Choulant.)

At the top in the center of the sheet is a cut representing a tiled bath-room. Above, a small window through which one sees the landscape. In the room sits a nude man on a wooden bench, and at his left sits a nude woman. Both have a narrow garment fastened at the hips. The woman's garment covers the right forearm. The man is holding in his left hand a water basin. The woman has in her hand a small tablet on which are inscribed the words, "Nosce te ipsium."

The veins of the arms and feet are drawn only on the male figure and marked with letters. The anterior wall of the trunk can be turned up, showing the anatomy, which is pre-Vesalian. On the footrest of the bench we find the initials R. S., and below a dissecting knife.

Above the male figure are the words, "Interiorum Corporis Humani Partium Viva delineatio." And above the female figure the words, "Pertuilis Anatomes Interiorum mulierbriis partium cognitio." On both sides of the plate are anatomic explanations, and below the whole width of the plate is taken up with directions for venesection and the names of the veins involved. The wood-cut is illuminated in colors. The name Vesalius on the title-page is a misnomer, as the anatomy is distinctly pre-Vesalian, and poor pre-Vesalian anatomy at that.

Variant A: Same title. Letter-press in French; 1613. Folio.

A second edition was printed in Paris by Michel de Mathoniere, 1613.

TYPE VI.—SINGLE SHEET

Viscerum hoc est interiorum corporis humani partium descriptio. N. D. (Choulant.)

An oblong folio sheet, consisting of several prints pasted together. The two central sheets show in one a nude man seated, and in the other a nude woman seated. Each figure has six or seven flaps, which are cut out and fastened one on top of the other in such a way as to be folded aside, giving a view of the internal organs. All the parts are marked with letters, except those of the female genitalia, which are marked by numbers. To the left of the man is a shield with the words:

"Antwerpiae, apud Sylvestrum Parsium, Typographium." Both figures are well drawn and well engraved. Sylvester de Paris was a form-cutter and printed epistles in Antwerp during the first half of the sixteenth century.

CLASS FOUR.—THE FEMALE FIGURE ALONE

TYPE I.—TWO SHEETS

Tabulae de matrice mulierum et impregnatione (1520). Double folio sheet; on the left folio, the figure; the right, the text in Latin. 27 x 36 cm.***

A hitherto undescribed gynecological anatomical plate, probably unique. There is represented with coarse wood-cut technic a squatting figure of a woman with the abdomen laid open, showing the genitalia. On the lower-side margins in Latin type are initial letters which direct attention to the separate parts of the body, which are also used for the explanation on the accompanying leaf. This is, of course, a direct copy of the Ketam figure, and probably an enlargement of the same wood-cut figure which was used in *Eyn Gert Artzney die hoc noch steet*, 1525.

While this plate is definitely derived from the celebrated Ketam illustration, it is impossible to determine whether it antedated or succeeded the illustration in the book quoted above, which was published in 1525. The dealers gave the date about 1520, but I am inclined to place it about 1530.

TYPE II.—SINGLE SHEET

Anathomia oder abconterfectung eines Weybsleyb, wie er inwendig gestaltet ist. Jobst de Negker, Augsburg, 1538. Letter-press in Latin. (Weindler.*)

This sheet has been studied by Wickerscheimer (*Bulletin de la Societe Francaise d'Histoire de la Medecine*, April, 1921). Weindler prints a copy of this plate, which was in the Klein collection, now transferred to the Library at Louvain. Both authors agree that no companion plate is known, so it must be considered in this classification. At any rate, it is probably the precursor of the Vogtherr Eve and may even indeed be a part of the first Adam and Eve set.

TYPE III.—SINGLE SHEET

Pertutilis Anatomes Interierum Muileris Partium Cogniti, ac earnndem sitis figura, numerus Positio hvd iniucunda Cognitu. Paris, Jean Roulle, 1540. Letter-press in Latin. 43 x 32 cm. (Schwarz.)

This plate has just been described in the catalogue of Dr. Ignaz Schwarz, Vienna. It shows the female figure seated, with the thoracic abdominal cavities open and the individual organs represented by superimposed flaps.

TYPE IV.—SINGLE SHEET

Della Parti Donna che Servona alla Generatione. Venetia, 1611. Letter-press in Italian. The sheet itself measures 40 x 29 cm.; the figure alone 32 x 18 cm.***

This sheet represents a woman seated, with a drapery thrown over the knees and the right hand resting upon a beautiful vase. It is probably a foundation sheet upon which have been superimposed flaps, but there is no evidence that flaps were ever used upon this particular sheet. The letter-press is in Italian; on the left hand there is a decorated initial letter with Latin type, and on the right hand a historiated initial letter and Latin type. Surrounding the whole a beautiful decorative border. This particular sheet has not been described, as far as I am able to determine.

TYPE V.—SINGLE SHEET

Authorizzare da Claffici Dottori. Venetia, 1683. The entire sheet measures 56.5 x 45 cm.; figure alone, 33 x 19.5 cm. Letter-press in Italian. (St. Mark's Library, Venice.**)

On the inside of the back cover of the famous "Tabulae," in St. Mark's Library, Venice, is pasted a fugitive sheet with the above title. The entire sheet has dimensions given above, and it is a definite copy of the female figure of Remmelin, but, as so frequently happens in copies, the figures are reversed as to right and left. The left foot of the female reposes upon a pedestal in this sheet, whereas in the original Remmelin the right foot reposes upon a skull. The snake is first brought to attention in the edition of Gemminis, 1545. The apple must always be present, so in the Remmelin figure it is in the snake's mouth. There are two subsidiary figures in this sheet. These figures are to be seen in Remmelin. There are six columns of letter-press, with the frequent use of italics, the whole surrounded by a decorative border.

CLASS FIVE.—THE MALE FIGURE ALONE

SINGLE SHEET

Tabula exhibens insigniora, maris viscera. Viitebergae, Barth. Schonbornio. N. D. The sheet is 42 x 32.5 cm. The figure in the upper

left-hand corner is 18 x 32.5 cm., surrounded by a black border. A decorative wood-cut border surrounds the entire sheet. Letter-press in Latin, with many words and phrases in Greek.***

The main figure has the features of Vesalius; the hair-line is continuous with the beard and the upper lip is covered with a mustache. A cloth printed in yellow covers the thighs. There are ten figures, designated by the letters A to H, surrounding the main figure. There are three superimposed flaps.

Variant A: Same title. (De Lint.**)

Dr. De Lint gives this sheet as printed at Viiteberg, edited by D. Barth. Schonbornio, about 1590. In this particular plate the hair-line is quite like that of the portrait of Vesalius in the *Fabrica*. The beard, however, does not go in front of the ears and the upper lip is bare.

PROCEEDINGS

OF THE

SECTION OF PUBLIC HEALTH AND INDUSTRIAL MEDICINE

MAY 18

Industrial Dusts of Pennsylvania and a Program of Study[1]

BY JAMES FREDERICK ROGERS, M.D.,
ACTING ASSISTANT SURGEON U. S. PUBLIC HEALTH SERVICE

The piece of work which the Division of Industrial Hygiene and Sanitation of the United States Public Health Service has undertaken in Pennsylvania is still in a preliminary stage, and we will be most glad of any suggestions which you may offer in helping us to shape our program.

As you are doubtless aware, this division of the Public Health Service, for a number of years, has, among other services to industry, been conducting investigations of health conditions in various occupations—in the garment trade, the potteries, the iron and steel mills, the chemical industries, the brass foundries, in a tobacco factory, in the glass industry, in the cement industry, etc. The results of some of these surveys have been published.

Dr. L. R. Thompson, the medical officer in charge of the office of Industrial Hygiene and Sanitation, a sanitarian of wide experience, has planned a thoroughgoing study of the more important industrial dusts. Pennsylvania, through its public officials, has

[1] Approved for publication by the Surgeon-General.

offered itself as a general field for such work, and a number of dusty trades are represented on a considerable scale within its boundaries.

The milling industry is a large one, and I believe no thorough study of the effects of working in the dust of flour mills has yet been made. There are many cotton and silk mills, and the morbidity in these trades is said to be due to a large extent to dust. There are many planing mills and the study of the effects of wood dust has been somewhat neglected. About one-third of the cement of the country is produced in Pennsylvania; there are numerous grinding shops for various metals; there are many tobacco shops; there are many slate and stone quarries. With the extent of these and other dusty industries in Pennsylvania, and the amount of risk they presumably entail, you are better acquainted than I am. Above everything else, the coal industry, as a dusty trade, seems preëminent, and anthracite dust is a nearly unique feature in this state. Moreover, a quarter of a million men are exposed to conditions inside anthracite coal mines. It is the anthracite coal industry which has been chosen for our present study.

The effects of working in coal-dust have interested sanitarians for a long time. The condition known as anthracosis was recognized over a hundred years ago, and, indeed, as someone has remarked, physicians were more interested in this disease then than they are now, because it was more frequent or in more exaggerated form. The low death-rate from tuberculosis among coal-miners has been long a source of speculation. However, the seeming beneficent action of coal-mining on this disease has tended to cause us to overlook its possible bad effects in causing the appearance of other respiratory diseases.

The special group of workers known as rock-miners, who do the tunneling from coal seam to coal seam, have not only no exemption from tuberculosis but very frequently meet an early death from this disease. All miners do a certain amount of rock work, and the amount of rock dust breathed may have much to do with relative morbidities.

That the coal-miner is subject to respiratory diseases other than

tuberculosis was noted in this state at least as far back as 1868. In the *Transactions of the Medical Society of Pennsylvania* of that year, Dr. J. T. Carpenter, of Schuylkill County, said: "The respiratory apparatus presents us with a very large percentage of cases of disease among miners. Bronchial irritations are continual and chronic bronchitis becomes often a constitutional affection attended by great debility. A peculiar asthmatic character of cough is generally noticed; emphysema is detected and the sputa are black. Miners' asthma is chronic bronchitis with thickening of the air-passages, emphysema and nervous distress in breathing. Acute pneumonia supervenes in many cases on some exposure, and is apt to prove fatal."

In a questionnaire study of the Geographical Distribution of Consumption of the Lungs and of Malarial Diseases, made in 1885 by Prof. Pepper, of the University of Pennsylvania, Dr. Corso, of Kingston, wrote: "Dust from anthracite induces a form of consumption in which asthma is a prominent symptom. Miners who cut rock tunnels," he observes, "suffer from a disease known as rock-miners' consumption, and resembling miliary tuberculosis. Rock-mining is considered more dangerous than coal-mining."

Dr. Matins, of Luzerne County, wrote: "I have known many persons, apparently in a decline, entirely cured by inhaling the dust of the mines and the smoke from lard-oil lamps and burning powder and drinking the water impregnated with sulphates." Notwithstanding these cures for consumption, he mentions that miners' asthma is frequent.

Much laboratory work has been done upon the effects of coal-dust upon the lungs, but with somewhat conflicting results or interpretations of results. The study of this subject by Wainwright and Nichols in 1905 was excellent, and it is to be regretted that these men have not found opportunity for further work along this line. The sickness survey among miners conducted by the Metropolitan Life Insurance Company was also a suggestive piece of work. Aside from these, I believe, no really thoroughgoing study of the subject has been conducted.

A study was planned by Dr. Landis, of the Phipps Institute, together with Dr. Charles H. Miner, of Wilkes-Barre (now State Commissioner of Health), Dr. Smythe, Dr. Mengel and Dr. Wainwright; but when they learned that the Public Health Service would undertake the work, they very generously withdrew from the field.

Our plan of attack is, in general, to study on the spot (*a*) the conditions under which the miners work, and (*b*) the men themselves, to discover to what extent they exhibit anything which may be attributed to the peculiar conditions of labor to which they are exposed.

1. For the investigation of working conditions we will send a trained sanitarian into the mines to study the character of the dust, the size, shape and number of particles breathed, the admixtures of stone-dust, etc. He will study the gases, the humidity, temperatures and air movement. He will also observe the men at their work, their posture and occupational methods, as well as their sanitary or insanitary habits.

2. In determining the effect of these conditions upon the health of the miner, our plan is to select a group of 1000 men working inside the mines and representing the different age groups. All absences from work of these men will be reported, and a special nurse will visit the home of each absentee to determine the cause of the absence, and if from sickness, the nature of the illness. These sickness records will be carried out for a period of at least two years, and the results will be compared with such records from other industries.

3. We will endeavor to make thorough physical examinations, with a careful occupational history of as many of this 1000 men as we can persuade to consent to examination: First, to secure general statistical data, and second, for the discovery of pathological conditions incident to occupational exposure.

4. *X*-ray pictures will be taken of the chests of representative groups of these miners at the different age periods and for different occupations in the mine.

5. Should any of these men be killed or die of sickness during

the course of this study, an effort will be made to secure autopsies and to make studies of the lungs.

6. Observations will be made by our absentee visitor of the conditions under which the miners of the group live.

7. A study will be made of cases of anthracosis, miners' asthma and other diseases of miners in clinics or hospitals.

8. The vital statistics of miners of the anthracite region will be collected and tabulated.

When this ground has been covered there is need, of course, of the correlation of all the facts learned about mine conditions with the facts about sickness and disability in the mines.

Arlidge remarked some thirty years ago that the severe types of anthracosis in England had nearly disappeared under modern methods of ventilation, and it may be that respiratory affections are not so common among miners in this country as is supposed. Granting, however, that coal-dust or other conditions in mines are found to be the cause of diseases of the respiratory organs exceeding the normal, it becomes an economic problem to determine the minimum of dust which produces such results. We city dwellers are all more or less anthracotic and the lungs of any middle-aged resident of the coal region are as black as a piece of coal. There must be some quantitative point where the breathing of coal dust ceases to be innocuous.

Such an investigation as this is easier planned than carried out. From past experiences we realize some of the difficulties ahead. Leisurely working in a laboratory, with rabbits or guinea-pigs, is simple compared with dealing with humans, indifferent or lukewarm as to their physical welfare. A thousand animals are more easily worked with than one man. As the Miners' Phthisis Prevention Committee of South Africa say in their final report: "We have not yet got to the stage that industrial trouble ensues when conditions underground become unhealthy. Apparently, the question of a slight increase in pay is still looked upon as much more important than one affecting the health of all concerned." However, changes are coming, and it is to be noted that industrial diseases were included in the Joyce Compensation Bill introduced in the present legislature.

If there are any other means of attacking the problem, or changes in our plan which you would advise, we will be very glad indeed to know them.

DISCUSSION

Dr. Elizabeth B. Bricker: I think I can voice the opinion of everyone here when I say that we have been not only very much interested in Dr. Rogers' paper, but that we have been very much enlightened, and that we will look forward with great interest to hearing the final report of the work of the United States Public Health Service in the anthracite regions of Pennsylvania. What I should like to say in my capacity as one of the medical inspectors in the Pennsylvania Department of Labor and Industry, is simply to call attention to the relation of that department to the dusty trades, and especially to what we can do to protect the worker in these trades as much as possible.

There are three main ways of protecting the worker: The first is to prevent dust suspension. This has been accomplished in quite a few industries, the most notable example being the use of the water drill in mining. The next way is to prevent the worker from inhaling suspended dust, and that is usually accomplished by having him wear a respirator, provided you can get him to do so. That is often quite difficult, and possibly with good reason. Take, for instance, the caustic worker who may be provided by the employer with a well-fitting respirator: He may wear it on a hot day when he is perspiring freely, and along the edges of the respirator the caustic dust will settle and he will have a caustic burn of the face. It will take some persuasion to make that man wear that respirator again under similar conditions. A simple respirator that might be substituted for the more elaborate one, and useful for many kinds of work, is one which is recommended by the State Department of Labor of New York. Take a piece of muslin 33 inches long and 5 inches wide. A little to each side of the center, along one edge, cut two rather shallow arcs of circles. Then lay the muslin down and over the center place absorbent cotton $\frac{3}{4}$ inch thick, 5 inches wide and 6 inches long. Cover the cotton with a piece of gauze to keep it firm, and with two safety-pins fasten the whole to the muslin. This is tied over the man's face and makes a very efficient respirator. It is light in weight, absorbs moisture, prevents dust from being inhaled, is easy to adjust, and at the end of the day may be washed or discarded as the worker or his employer decides. The third point of attack against dust is the removal of all dust at its point of origin. The Departments of Labor of New York, New Jersey and Pennsylvania have formulated for this purpose standards which are

somewhat similar in character. They cover three principal points: First, the size, construction and location of the hood over the point of operation where the dust is being generated; second, the size, location and installation of ducts leading from the hoods to the point where the dust is collected; third, the amount of suction to be maintained throughout this system.

I do not know on what basis these standards were originally formulated, but I do know that it is just such studies as Dr. Rogers and the United States Public Health Service have made in the past and are making now that will enable departments like ours, in the future, to revise our present standards and to make new standards on a definite, accurate, scientific basis.

Dr. T. Grier Miller: I, too, feel that such studies as Dr. Rogers is contemplating are going to be of the very greatest importance to us. It seems to me that we must all appreciate that there is a great deal of difference in the kinds of dusts, especially as to their effects in producing pulmonary disease. So far as I know there is no evidence to show that organic dust leads to pulmonary fibrosis. Even among the inorganic dusts there is a great difference insofar as the pulmonary affections are concerned. Dr. Smyth and I have made some studies on dust in numerous industries and we found that the cement industry was by far the most dusty of those we encountered. We found something like 13,000,000 dust particles per cubic foot, whereas in ordinary city air we found only about 50,000; yet when Dr. Pancoast, Dr. Landis and I studied these cement workers by *x*-ray and by physical examination we found very little. I think only one had even a cough. On physical examination very little was found and the *x*-ray studies showed only a moderate amount of fibrosis as compared with the marked fibrosis which we found in certain other workers. The explanation lies in the small amount of silica in cement dust. The dusts that seemed to give rise to most trouble were those which contained considerable quantities of silica.

Steel-grinders and flint-workers very readily develop pulmonary fibrosis, and we found that men who had worked only five to ten years in this industry were coughing and had expectoration, and when they were studied under the *x*-ray they showed marked pulmonary fibrosis. This, of course, is quite in line with the findings of the English, particularly the work done in certain gold mines in Africa where there is a great quantity of silica in the dust.

Even in the inorganic dusts the effects on the individuals are suprisingly mild in the early years of their association with the industry. So far as symptoms are concerned, most of these people get along without any for

ten, fifteen and twenty years, and in the case of potters we found for thirty and forty years, without any very definite symptoms; and when they finally developed the first was usually a cough and later dyspnea. After a while mucoid or mucopurulent expectoration and even hemoptysis may develop when there is really no tuberculosis. On examination, persons markedly affected will show some limitation in expansion of the chest and sometimes deformities, and occasionally the heart may be displaced, and on auscultation all sorts of rales are likely to be heard. In the early stages of the pulmonary disease there are no physical signs, but in this stage it is possible to determine the condition by *x*-ray, the latter showing some thickening of the shadows over the hilum of the lungs, later a mottled appearance of the entire pulmonary structure, and, finally, in what most *x*-ray men term the third stage, one gets very pronounced mottling with dense areas. The parts of the lungs most definitely affected by the dust are the middle portions of the lungs, not especially the apices, and more particularly the posterior portions of the lungs rather than the anterior.

As to whether or not tuberculosis is produced by dust, of course we can say, No! Tuberculosis is not produced by dust; tuberculosis is produced by the tubercle bacillus and nothing else. The thing that dust produces is pulmonary fibrosis. I feel that this ought to be emphasized, because there is a lot of confusion about it, although a very simple thing. Whether or not pulmonary fibrosis predisposes to tuberculosis is the unsettled question. There are those who feel that pulmonary fibrosis is rather a protection against tuberculosis. Dr. Landis particularly has taken that point of view, and has shown, in potters at least, that while the tuberculosis death-rate is rather high, it occurs at a later age than in the general population. It appears, therefore, that if a person has pulmonary fibrosis and tuberculosis, the latter process is not so rapid as in those without fibrosis.

Dr. Rogers says that for a long time it has been thought that tuberculosis was not particularly frequent among coal-miners, and once there was a theory that the coal acted in a germicidal way; but I do not believe anyone now feels that that is true. Some of the English authors rather have the feeling that the comparative harmlessness of coal-dust is because the coal-dust particles which get into the lung epithelium are so irritating that the cells are thrown off by a catarrhal reaction and finally expectorated; whereas silica dust, being less irritating, does not set up such a reaction, many of the particles going deeper into the lymphatics and eventually leading to fibrosis.

Dr. Henry Field Smyth: This question of industrial dust and its effects is one in which I have been personally very much interested for

some time. I am delighted that this survey that has been talked of for a year or more is at last under way and I sincerely trust that nothing will prevent its being pushed to completion. I have been very much interested in the question of the methods of estimation of dust. I would like to ask Dr. Rogers what methods they propose to use in determining the size of the dust particles and in counting the dust? I trust that if they have occasion, as no doubt they will, to conduct postmortems on any of these miners, they will repeat the experiments made by the South African investigators as to the determination of the size of dust particles lodging in the lungs. I think this is an important matter; to determine just what is the danger-point, what are the most dangerous particles. The work in South Africa indicates that the majority of particles lodging in lungs are from $\frac{1}{2}$ to 2 microns in diameter. Yet the critics of the Palmer apparatus for estimating industrial dust are condemning the apparatus because it does not entrain enough of the so-called ultramicroscopic particles, the particles under $\frac{1}{4}$ micron in diamter. I did a little work in examining the lungs of guinea-pigs that had been dusted with silicious dusts, and in counting the particles, using the same methods they used in South Africa, I found practically the same relative distribution, with no evidence to any extent of accretions of ultramicroscopic dust particles. Personally, I do not believe that the very minute dust particles that remain in suspension in still air (particles, $\frac{1}{10}$ micron or under) are dangerous unless they are toxic in nature. Toxic dust is dangerous no matter what its size. I hope they will make determinations, if they have the opportunity, on the size of the dust particles. I think, also, the study should be accompanied by laboratory experiments on animals to determine the relative effect on tuberculous infection of the silicious dusts, the coal dust and calcium dust. It has been claimed not only that coal is beneficial but that calcium is even more beneficial. They sometimes say they can cure individuals with tuberculosis by putting them to work in a limestone quarry. I am rather skeptical about that myself, but I think the different types of dust should be tested by animal experimentation. I have done considerable of this work myself, just enough to make me feel that it has distinct value, and I hope that they will see their way clear to parallel their investigations by working investigations, including animal experimentation.

Dr. Rogers (closing): I want to thank those who have given us hints of what we are going to do. We have not planned as yet on laboratory work, but, as Dr. Smyth says, a great deal has been done, but nothing very definite, and it ought to be a part of the complete program in this study. Our sanitarian for dust work has not begun work yet, but he

fancies the Farmer, or modified Farmer, that is now being studied with those of Winslow and Weaver. I know the Farmer machine is not altogether satisfactory. I had some experience in the optical shop with carborundum. My tests were made chiefly with carborundum. The machine will not take up the larger particles—hardly pick them up at all —and yet the fine particles were run through. Our chemist, who is taking these samples in cement factories in Hagerstown, employed four Farmer machines together, run at a standard rate, and found that he got a considerable amount of the dust in the four machines, but that he did not get the total quantity in the four combined. We probably shall use modified Farmer machines.

Industrial Anthrax in Pennsylvania

BY HENRY FIELD SMYTH, M.D.

The question of industrial anthrax in Pennsylvania is one in which I became interested through the instrumentality of our State Department of Labor, and I have been working for the last three or four years on this question, obtaining some very interesting and very instructive data.

In the first place, we are apt to think of anthrax as a disease prevalent in some other country, not realizing its existence in endemic foci in this country, and feeling that where we have a case of this disease it is always imported. We always look for the cause in that particular case in the handling of some imported material. Usually it is there, but not always. I was interested in collecting statistics of anthrax morbidity and mortality in this country for some years back. This mortality the census lists under two heads, urban and rural. In looking over rural anthrax reported in this special Anthrax Bulletin of the Bureau of Labor Statistics, we find a number of cases with their histories recorded with absolutely no suggestion that the case was contracted by handling any foreign material. For example, in one instance out on the plains, a ranchman had several cattle die with a curious bloated condition of the abdomen, and he

wondered what was the matter, and cut one open to find out. His is one of the cases listed in the mortality statistics. Two or three years ago, down in Louisiana, a farmer had a mule die, and to salvage what he could, he and his son skinned the mule and sold the hide. Forty-eight hours afterward they both contracted anthrax. Fortunately, they are listed under the morbidity and not the mortality statistics. These are just instances. Now I have found such instances of distinctly rural anthrax in 28 states, 120 cases from 1910 to 1920. In the first column are cases up to 1918. You see how many states are listed in that column (pointing to the chart). In 1918 the country began to be flooded with cheap shaving brushes made from imported China horsehair often infected with anthrax, which might explain some of the so-called rural anthrax later. These cheap horsehair shaving brushes were not put on the market until then. So much for rural anthrax and the fact that we must bear in mind that while at present we have comparatively little domestic anthrax, yet it is a menace and a widespread menace.

Anthrax in industry is associated particularly with three industries: wool, horsehair, and skins and hides, and the classical textbook internal anthrax is woolsorters' disease or pulmonary anthrax. Fortunately, we know very little about that in this country. We do not get the lower grade of wool, unwashed dead wool clipped from dead animals. Most of our wool is clipped from healthy live animals. We occasionally get cases of anthrax in wool, but very seldom. I had been able to find up until 1922 but 3 cases in Pennsylvania definitely attributed to wool, when I made this chart. I have heard of 2 other cases within the last year in Philadelphia treated in one of our Philadelphia hospitals.

The next important industry is the one in which I first started investigating work in connection with Dr. Bricker for the State Department of Labor—the horsehair industry. This includes the dressing, combing, hackling and sorting of horsehair that it may be made into horsehair cloth, curled hair or brushes. We have had 10 cases of anthrax from horsehair, 3 in 1912, 1 in 1917, 1 in 1918, 1 in 1920 and 4 in 1922 in this city.

All of these cases, so far as we know, came from handling imported hair. I have made cultures in one or two instances from domestic hair organisms, which, aside from the fact that they would not kill laboratory animals, would be called anthrax, but I have no case on record of definitely proved anthrax from domestic hair. A great deal of the hair is imported unwashed and a great deal of hair imported from China is dressed without washing. The majority of that hair contains anthrax. In New York it was found that 75 per cent of hair shipments from China contained anthrax. It was from that hair that the cheap horsehair shaving brushes were made that sold for 10 or 15 cents. I have cultured virulent anthrax from such hair bought in a shop in this city.

Our great anthrax menace in Pennsylvania is in the tanning industry, in the handling of goat skins and cattle and horse hides. I have separated the two industries in my tables because of the different sources of the raw material and the differences in the way in which it is handled. Until comparatively recently a great many of the cattle hides imported were imported just as the goat skins are, being gathered by small collecting agents throughout the country, bundled together and brought to central points, rebundled and shipped in bulk. The majority of these shipments contained hides with anthrax. At present the bulk of the cattle hides tanned in this state are from healthy packer-killed cattle; therefore the anthrax is not as great a menace in the cattle hide tanneries as it used to be. It is far different in the tanning of goat skins. Anthrax is a menace here and is likely to be so for some years to come, unless entirely different methods are introduced. In the years 1910 to 1922 we have had in this state 74 cases of anthrax, with 16 deaths in the handling of cattle hides; but in 1920, 1921 and 1922 there were comparatively few cases, because the packers controlled not only the cattle industry in this country but in South America, and they also controlled the tanneries making sole leather and using cattle hide. They at present are able to supply hides for most of their tanneries. There are a few tanneries which are tanning hides not from packer-killed cattle and in three of these we found anthrax.

There are fewer goat-skin tanneries in this state, most of them in the limits of this city. There were 61 cases with 9 deaths in goat-skin tanneries from 1910 to 1922. You will see that the goat-skin risk continues, 19 cases being recorded in 1921, and I believe there have been several in 1923. My statistics do not go into 1923. Practically all the goat skins are imported from countries where anthrax is prevalent, Russia, China, India, South America and Mexico, and from all these countries we have found hides containing virulent anthrax organisms. All these countries have a history of supplying hides which were the cause of workers contracting anthrax. At the time we made the tannery survey of this State, Dr. Bricker and I visited every one but two; one did not want us, and one was in a little out-of-the-way place, where I think the father and son made up the whole force of the tannery. We visited over 70 tanneries, and we found that there were between 7000 and 8000 employees in these tanneries. Now not every tannery employee is exposed to anthrax. In my statistical work I have separated them into those directly exposed and those only indirectly exposed. Those who put to soak and handle the hides until the time they are ready to go into the tanning liquor (almost 8 per cent of these employees) are directly exposed. There were 1040 of these. In the five years where there were no unusual labor or trade conditions to affect the industry, we find a yearly average of 1.8 per cent contracting anthrax, a continuing menace in the cattle-hide industries. In the goat-skin industries the percentage was not quite, but almost as high, an average of $1\frac{3}{4}$ per cent of these employees contracted anthrax each year. The next table shows the results of search for organisms in hides, dust or soap liquors. In all tanneries 15 per cent yielded positive anthrax cultures. Only three of the cattle-hide tanneries, or 6 per cent, but 54 per cent of the goat-skin tanneries, had virulent organisms in the vats or bins at the time we made the survey.

As part of our statistical work we tabulated the results of treatment methods. You will see a good deal written on the question of the use of anthrax serum: some men are claiming that that is the only treatment worth while, in fact, that it is practically criminal

to touch the anthrax pustule in any way. The nearest approach to local treatment recommended by them is simply to inject anthrax serum around the lesion, also using the serum intravenously. Now every case of localized anthrax is likely to become a case of generalized anthrax sooner or later.

The physician who had treated more cases of anthrax at the time of survey than any other man in the state does not use anthrax serum at all. The serum is used with very good results in this state, and I approve of its use; but I take issue with the men who say never do anything else, and who attempt to convey the idea that there is nothing in specific anthrax therapy but the use of serum. I have found the principal methods of treatment used in Pennsylvania to be (*a*) the use of serum alone, (*b*) a combination of serum and excision of the lesion, (*c*) the use of excision with 25 to 50 per cent of phenol injections around the lesion, and (*d*) in one hospital phenol injections alone. As to results of treatment: in the 5 cases where serum alone was used, 3 proved fatal. Thirty-seven cases were treated with serum and excision, with a mortality of 13.5 per cent, and that is about the mortality in this state up to the present. Where phenol was used, whether with excision or without, the mortality was 10.9 per cent. In all cases where excision was used the mortality was 11.6 per cent; and where serum was used, 20 per cent. The highest mortality occurred where there was least attempt at local interference. Simple incision is worse than nothing, but free excision, our statistics seemed to prove, is of distinct value, and I should say it was certainly of value to the man who could not get serum. These statistical records show that the use of phenol has decided value. Personally, I would want serum, but I would also want the anthrax pustule excised, and if serum were not available, I certainly would want phenol injected around the wound.

Another light upon the value of treatment methods is given by the tabulation of the time lost of non-fatal cases. You will note that where phenol was used the average time lost was less than where no phenol was used. Now in judging these statistics, you must also bear in mind this fact: the men who are using phenol

are those who are working for the cattle-hide industry, and they are often located far away from the tannery. Many of their patients must be transported for diagnosis and treatment 50 to 100 miles to the physician, often over rough mountain roads. With anthrax, as with most every severe fulminating infection, the sooner treatment is started the better. This makes the results of those who are not using serum remarkable.

I want to say a few words as to methods of prevention: The United States Government Bureau of Animal Industry attempts to handle this question, but handles it in a very unscientific and unsatisfactory manner. In the horsehair industry they say that horsehair shall be subject to disinfection unless it comes (and the same thing applies to skins and hides, unless they come) from countries where they can be accompanied by a consular certificate stating that the region from which they are collected is free from anthrax. That certificate is not worth the paper it is written on. In Massachusetts the state authorities have cultured anthrax from so-called certified skins and so have we in Pennsylvania. Now as to disinfection of goods that come in without certificate: horsehair is permitted to be treated by exposing for fifteen minutes to 200° F., an absolutely absurd provision which could not destroy spores. Horsehair can be disinfected adequately and simply in an autoclave. For the cattle hides and goat skins they say that these non-certified skins and hides must be treated in one of the following ways: soak in either quicklime or calcium hydrate (they give you the choice) for twelve hours, in an emulsion containing 15 pounds of lime per 100 gallons, supposedly to make a 5 per cent of calcium oxide. As a matter of fact, it makes a little less than 2 per cent, provided your lime is pure, which it never is. I have cultured anthrax from skins after a seventeen-day soak in this lime suspension. Goat-skin manufacturers use it because Uncle Sam permits it. The Bureau also recommends other methods which will disinfect, but the tanner will not use them, saying they injure the leather. In Massachusetts they make leather routinely by these methods and obtain a fine product, but Pennsylvania tanners maintain they cannot. In the cattle-hide industry tanners

attempt to sterilize, and the Government allows them to, by soaking in a plain 1 to 1000 bichloride solution for twelve hours. They might as well soak in rain water. I have cultured virulent anthrax from a tannery in the northern part of this state, from a vat in which Indian buffalo hides had been soaking in 1 to 1000 bichloride twenty-four hours. We have elaborated a method in the laboratory which will kill anthrax and give beautifully tanned skins. Unfortunately, that method is very expensive at present. There are possibilities, however, of reducing the expense very materially. The method has been submitted to the Tanners' Council. A number of the tanners were very much interested in it, but the President of the Local Division, a tanner who had not had an anthrax case in his factory for a good many years was not willing to try it. I think this method will be tried out in another winter in one of our large Philadelphia tanneries.

DISCUSSION

Dr. Elizabeth B. Bricker: I have very little to say, except to corroborate in general what Dr. Smyth has said, because most of the studies the Pennsylvania Department of Labor and Industry has made on the subject have been carried on in conjunction with Dr. Smyth. I should, however, like to say one thing about his mortality statistics, which I think are too high. I have not discussed this with him, but I believe the statistics we got at the time that we made the survey were not the really true morbidity statistics. They were, I believe, good mortality statistics, but I feel sure there must have been a great many cases of anthrax that recovered which were not included in our list, and which, if included, would have brought down the mortality figure. Statistics for the state last year were considerably lower than those Dr. Smyth has given us.

I should like, at this time, to voice the appreciation of our department for the splendid coöperation we have received from the Philadelphia Hospital for Contagious Diseases in giving us very valuable statistics on the cases of anthrax occurring in Philadelphia. Our present Commissioner, Dr. Royal Meeker, is most anxious to get accurate statistics on this subject and Dr. Woody has been very kind in furnishing us with all the details we have requested from his records.

Dr. Woody: My acquaintance with anthrax begins after the patient reaches the hospital, and is, therefore, more the experience of the medical practitioner than that of the research worker or public health official. In the more than fifteen years of my connection with the Philadelphia Hospital for Contagious Diseases we have taken into the institution and treated 114 cases of anthrax. Of these there were 13 deaths, a mortality of 11.40 per cent. Dividing this period into two parts, of ten years and five years, will give some figures that are quite interesting. For the first ten years there were 56 cases with 11 deaths, a mortality of 19.64 per cent; for the last five years there were 58 cases with 2 deaths, a mortality of 3.70 per cent.

These figures show a greater number of admissions and a much lower death-rate (one-fifth) for the last five years than for the previous ten years. A further study will show that there were 18 cases admitted for 1922, and 15 were admitted for a little more than five months of 1923. The cause of the very low death-rate for the last five years is due to improved methods in prophylaxis and treatment. The educational work both among the public and the profession has saved undoubtedly many lives, for it is quite noticeable that the cases admitted in recent years are milder than those formerly admitted. Familiarity with the disease has meant early recognition and prompt treatment. The improvement in treatment can be traced to larger doses of the serum and to a change in the method of administering it. Formerly, the serum was given subcutaneously or intramuscularly, and in dosage recommended by the manufacturer. It is now given intravenously in all cases and in very large dosage, 100 cc to 150 cc at a time, and repeated as often as thought necessary, every six, eight or twelve hours. In some instances a total of 1000 cc has been given in a single case. Our experience with this serum has been the same as that with diphtheria, tetanus and other sera, in that the dosage to bring results must be greatly in excess of what is recommended by the manufacturer and the medical profession in general.

Surgery also plays an important role in the treatment of anthrax. Where the lesion is localized, accessible for operation and not too extensive in area, complete excision of the lesion together with the area of induration should be done. My experience is that practically all such cases get well.

The practice of administering serum intravenously and injecting it about the lesion has been strongly recommended by Regan and others. We have used it this way in a few cases and always with good results. I am glad to hear Dr. Smyth say that surgery in the treatment of anthrax should not be discarded. My feeling is that surgery is of equal or possibly of more importance than the serum in the mild or early cases where

the lesion is distinctly localized. This I know, for there have been times when serum has not been available and surgery was the only method employed.

Finally, let me say that my work with anthrax has led me to hope, and even to believe, that we might be able to say of this disease, as we have been able to say for some years about diphtheria, that given the cases within twenty-four hours of onset and administering prompt and vigorous treatment will give us a cure in every case.

Dr. Walter S. Cornell: Dr. Smyth has mentioned the interesting fact that the goat-skin tanneries examined by him were more heavily infested with anthrax than the cattle-skin tanneries, notwithstanding that there are more cases of anthrax among the cattle-skin workers than among goat-skin workers. Possibly the location of the malignant pustule in the case of goat-skin workers and cattle-skin workers respectively might furnish a clue to the reason for this apparent difference in risk.

Dr. Henry Field Smyth: Our records show the higher morbidity and mortality, going back in the entire period of ten or eleven years, in the cattle-hide tanneries. If you look at the records you will notice that the case-rate dropped off very much in cattle-hide tanneries, 1920, 1921 and 1922, whereas in the goat-skin tanneries it continued high. That was the period during which we made our survey. Most of the tanneries were not receiving any cattle hides that were not from fresh-killed animals, whereas goat-skin tanneries were still getting a majority of such hides which were liable to have anthrax spores on them. Where we cultured anthrax from cattle-hide tanneries, these tanneries had recently received hides that were not from packer-killed animals. Whenever that is the case you will get anthrax sooner or later. I was interested in Dr. Bricker's statement in regard to her idea of our mortality statistics. It recalled to my mind how very hypercritical we both were when compiling them. We threw out several cases which I think were anthrax, but we could not prove it, and we really had some cases which I think we were justified in ruling out. That brings up the importance of laboratory diagnosis. One case at Newport, Pa., reported as anthrax never came in contact with untanned hide. The diagnosis was sent in from the hospital as anthrax. It was a fatal case. When we looked up the hospital record it was staphylococcus septicemia, but no record of anthrax. You get cases diagnosed as lead poisoning because the man worked in the neighborhood of a lead factory. As an interesting sidelight on this work, I wanted to have some experimental work done in a tannery. In the tannery they thought I was a nuisance, they did not think they wanted to go into the matter to

that extent until Dr. Woody had an opportunity of cutting out an anthrax pustule from the shoulder of a member of that firm. After that they could not do too much for us. Dr. Woody referred to two cases in housewives. One, I suppose, contracted anthrax while mending her husband's clothes, by pricking her finger, the other from washing her husband's overalls. Also, in 6 or 7 cases in carpenters, anthrax was contracted from repairing old soak vats. I would like to ask Dr. Woody what he thinks of the theory of those South American workers who believe in non-specific protein therapy and have finally come to the administration of peptone, which they claim accomplishes the same results.

Dr. Woody: In answer to Dr. Smyth's question about the use of bovine serum, I would say that our custom has been to depend upon the antianthrax serum and surgery. I know that South American workers have written a great deal about bovine serum, and they have presented some figures that are quite sensational. The serum we use is made in Philadelphia.

OCTOBER 19

Proposed Plans for a New Water Supply for the City of Philadelphia

INTRODUCTION BY A. C. ABBOTT, M.D.

About ten or twelve years ago there culminated an activity in this city that had been going on for some seven or eight years. The object of that activity was to remove Philadelphia, sanitarily speaking, from a position that might be called disgraceful to one that might be called fairly respectable, by the introduction of a purified water supply—the purification having been accomplished by the process of filtration.

When it was proposed to undertake the purification of our water supply by filtration there was a great deal of controversy; many doubted the practicability of the plan. However, as you are aware, filtration was adopted and the results of that filtration

are common knowledge: Typhoid fever and other intestinal infections having been reduced to almost the vanishing point.

In this connection it is proper to state that those who advocated purifying the existing supply fully realized that it could not answer for all time; they knew that the growth of the city and the multiplication of industries along the banks of both the Schuylkill and Delaware Rivers would necessitate, at some time in the future, a new supply. That time seems to have arrived. Those who are interested in the welfare of the city, and who have at their disposal data on which trustworthy opinion can be based, realize that under existing conditions the Schuylkill River is inadequate to meet the demands that are being made upon it, and that the cost of purifying the water of that stream is almost prohibitive. It is proposed, therefore, to adopt a plan that may reasonably be expected to meet all future requirements of the city and that may properly be looked upon as a plan for the installation of a genuine metropolitan supply. There has been a good deal of discussion of the merits of different available watersheds within a reasonable distance of the city as sources of supply. As yet no conclusion has been reached, and I think it would be advantageous for all citizens interested in this subject to be informed as to just what it is proposed to do. The cost of this project—no matter which plan is adopted—is going to be very great, and you will be called upon to vote upon a loan to meet the expense. It is plain, therefore, that you should know something of how the money is to be expended. In order that you may know just what is being discussed, I have invited the Director of Public Works of the city, Mr. Caven, to lay before you, concisely, this evening, the various plans.

Another situation that confronts the city is the inadequate and antiquated methods that are in use for the disposal of the city's wastes. Director Caven has made a number of important improvements, but he still feels there is room for a great deal more to be done in that direction. He will discuss, this evening, that subject also.

It gives me great pleasure to introduce to you Mr. Frank H. Caven, Director of Public Works of the City of Philadelphia.

BY FRANK H. CAVEN

DIRECTOR OF PUBLIC WORKS

I come here tonight, not as an engineer but as a layman, to discuss the water problem, and it will be my endeavor to make it plain to you all so that it will be possible for you to pass judgment and arrive at some conclusion as to what would be to the best interests of our citizens with reference to the improvement of the water supply.

The history of the Philadelphia water department for the past hundred years can be written in a series of battles for funds to secure an adequate supply of wholesome water. The growth of the city has been so persistent and rapid that the improvements and additions that have been made to the plants have been insufficient by the time they were completed. The greatest improvement to the Philadelphia water supply was the construction of the filter works, in 1909, which your chairman has referred to. These works transferred the pumpage of nearly 200,000,000 gallons of water from the Schuylkill River to the Delaware River at Torresdale, and provided filter plants having a capacity of 350,000,000 gallons per day, and I might say to you tonight that we are pumping daily 350,000,000 gallons at this time.

The effect upon the health of the community of supplying filtered water was speedily noticeable and the improvement has steadily continued. In the old days, when raw, unfiltered water was supplied, the deaths per year in Philadelphia from typhoid fever averaged 1000 and over. In 1922 the total deaths for the entire year were only 52. As a matter of fact, typhoid fever has been eliminated insofar as the water supply is concerned, and the few cases occurring are contracted from either carriers or from outside sources. The supply of filtered water has been, in the past ten years, the means of saving from death from typhoid fever of at least 10,000 persons. The number of typhoid fever cases this year, up to October 13, was 196 as against 295 for the same period of last

year. The deaths this year, so far, were only 18, or half of those of 1922. This is the lowest death-rate from typhoid ever experienced in Philadelphia. If this continues the death-rate for 1923 will be 0.00014, which will place Philadelphia in the front rank in the typhoid fever mortality table of the cities of the United States. So there can be no doubt in anyone's mind that the safe and sane thing to do is to filter all water used by a municipality, no matter from what source it comes. Water can be clear and sparkling, but still be carrying deadly typhoid fever germs, and any improvement to the present water supply must include filters.

We have now arrived at the point where the city is obliged to change its source of water supply. While the filters are producing clear and non-disease-carrying water, they do not nor can they be adapted to eliminate certain tastes and odors that occur at irregular periods, particularly in the Schuylkill River. It is unpleasant to know that you are drinking filtered and chemically purified sewage. The conditions in the Schuylkill River are such that it is practically impossible to stop the entrance of sewage and trade wastes, and the amount of such contamination will steadily increase. Those of you who are familiar with the Schuylkill River, and have gone along its banks, cannot help but agree that it is going to get worse and worse as the days and months and years go on. It is very thickly settled, and a great number of industrial establishments are there and continue to be placed there. This fact, taken together with the inadequate quantity, makes it necessary to abandon the Schuylkill as a source of supply. It is interesting to know that for relatively long periods during the summer months the entire flow of the Schuylkill is pumped. In other words, we are taking practically the entire flow of the Schuylkill River now, at drought times, to supply the water to the sections which the Roxborough, Belmont and Queen Lane pumping stations serve. It is pumped, filtered and supplied to Philadelphia for domestic use. The great northeast section, as well as the southern portion of west and south Philadelphia, are demanding an increased supply of water, so that, irrespective of the elimination of the Schuylkill River as a

supply which gives us bad tastes and odors, the city is also demanding an increase, and a very large increase, in the present supply of water.

There has been a proposition submitted which advocates the impounding of water in the Perkiomen district, starting with the Green Lane reservoir, in which 15,000,000,000 gallons in a watershed of about 700 miles are obtainable. With such a reservoir completed, in the event of a drought such as was experienced last fall, the sluice-gates could be opened and 100,000,000 to 150,000,000 gallons of water released daily for approximately three or four months, depending upon the volume, and, as stated, in this way the quantity of water reaching the pumping stations and the filter plants will be greatly increased. The plan further provides that, when the entire Green Lane and Tohican projects are carried through, there would be delivered to the city of Philadelphia 250,000,000 gallons of water per day, this being only 50,000,000 gallons more than is being pumped or used with the flow of the Schuylkill at the present time. And the experts all agree that it is very necessary for the city to provide at least 600,000,000 gallons of water to take care of the city's needs in the next fifty years.

The water stored in the Green Lane reservoir would, by this plan, flow down the present channel of the Perkiomen into the Schuylkill River, and then to the pumping station, with the result that you will not get a straight drink of clear Perkiomen water, but a mixed drink, or a cocktail, composed of one-third Perkiomen and two-thirds Schuylkill water, with Pottstown, Norristown and Conshohocken sewage—the whole concoction equaling anything offered by the present-day bootlegger.

This proposition, the Green Lane project, as offered at this time, is one that makes it necessary to use, as I stated here, the Perkiomen Creek, the Perkiomen emptying into the Schuylkill and flowing down the Schuylkill to our pumping stations. That is nothing more than bringing about a flushing and diluting of the present Schuylkill River. How much improvement can be brought about in the diluting I will leave to you to judge. Of course, in their final completion of the project, it is intended

to build an aqueduct. After all the dams are completed up there—the Tohican, Green Lane and Perkiomen—then the aqueduct would be built, and it would bring down to the city of Philadelphia, or to our pumping stations now along the banks of the Schuylkill, the water without having been contaminated by the Schuylkill water, but only increasing our daily output 50,000,000 gallons.

In lieu of the proposition just outlined, I want to submit to you a plan, not original with me, but one that has been thought out by engineers who have had a long experience in matters pertaining to Philadelphia's water supply. I might say, however, before taking up the proposition, that the cost of the Perkiomen system—let us call it that—will be $61,800,000, and the addition of the Tohican Creek would mean $14,600,000 more, making a total cost of $76,400,000, and it would take at the very shortest time, possibly, ten years to complete. The plan that I would like to discuss tonight provides for enough water, namely, 600,000,000 gallons, to take care of the city's growth in the next fifty years. And that for a sum of money less than for the plan of the Perkiomen, which only increases the water supply, as I have stated, 50,000,000 gallons.

The Neshaminy Creek: This creek has a watershed of 212 square miles, above the falls, which, with adequate storage, will furnish 170,000,000 gallons per day. The Delaware River, above Trenton, has a watershed of 6916 square miles. The average minimum flow of the river is over 12,000,000,000 gallons per day, the ordinary summer flow being about 2,000,000,000 gallons, and the remainder of the year it is from three to five times this amount. There is a record of the flow for a very short period, some years back, of 900,000,000 gallons per day—a supply ample for fifty years. 600,000,000 gallons per day could be obtained from the Neshaminy Creek and the Delaware River above Yardley. The cost of the Neshaminy-Delaware project will be $60,000,000, which includes the aqueducts to Queen Lane and Torresdale, with a total capacity of 700,000,000 gallons. These aqueducts will be built large enough, if called upon in case of an increase in

pumpage from the Neshaminy and Delaware River, to provide for an additional 100,000,000 gallons more than the 600,000,000 gallons we contemplate pumping, and the connections also to the existing filter stations.

The plan, briefly, is as follows: A pumping station on the Delaware River, above tide-water, equipped to pump 600,000,000 gallons per day; aqueducts from the Delaware River to the Neshaminy reservoir, with a capacity of 600,000,000 gallons per day; the building of a dam 80 feet high across Neshaminy valley district, and the forming of a storage reservoir of 10,000,000,000 gallons. This reservoir will provide ample storage and subsiding and equalizing basin, and enable full advantage to be taken of the storm-flow in the Neshaminy watershed, which will greatly reduce pumpage from the Delaware River. Calculation shows that for an average year there will be many days upon which it will be unnecessary to pump any water from the Delaware River, and the average yearly pumpage from the Delaware River, when the consumption per day is 600,000,000 gallons, will not exceed 350,000,000 daily. I state this because in Councils one day, in proposing this plan, one of the members raised the question and said that the city of Philadelphia would have to be careful with reference to pumping water from the Delaware because half of it belongs to New Jersey, or possibly New York State, and I want to show you that it is absurd, because the amount of water that Pennsylvania would take away for Philadelphia, or pump for its portion of the water, would be *nil* compared to the flow.

The water from the Neshaminy reservoir will be conveyed by an aqueduct to the Queen Lane pumping station. That is out on the Schuylkill just above the Falls. And I might say, tonight, that during my term of office that station has been remodelled and rebuilt, all the old reciprocating pumps have been abandoned, and where the station was capable of pumping only 60,000,000 to 70,000,000 gallons per day we now have four cross compound turbine engines or pumps capable of putting up, well, 160,000,000 gallons very readily. So there is no question that this station is up to date and will take care of a great deal more

pumpage than it can possibly get from the Schuylkill River. This plan provides that through the Neshaminy—from the Neshaminy Creek or the reservoir—the aqueduct will transfer the water over to Queen Lane. The Queen Lane aqueduct will be 17 feet in diameter, with a capacity of 700,000,000 gallons per day from the reservoir to its junction with the Torresdale aqueduct, which will be 13 feet in diameter, having a capacity of 350,000,000 gallons per day. In one of the slides I can show you that very plainly. The aqueduct starts from the Neshaminy reservoir and extends to the Queen Lane pumping stations. There is a branch taken out which goes to the Torresdale filters, so they will get their share of the necessary amount of water. The total length of the Queen Lane aqueduct will be 18½ miles, and the Torresdale 4½ miles. These aqueducts will be constructed 125 to 150 feet below the surface and lined with concrete, and two-thirds of the length will be under legally open streets. I quote that in order to show that there will be less expense in acquiring the right-of-way for these distances. The new supply will be delivered under from 30 to 50 feet head at the Queen Lane pumping station and Torresdale filtering plant.

From the Queen Lane station 100,000,000 gallons per day will be pumped to the Belmont filters for the West Philadelphia supply, and I state to you tonight that West Philadelphia is getting about 50,000,000 gallons per day, so that this has provided for a 100 per cent increase.

50,000,000 gallons will be pumped to the Roxborough filters for the Germantown and Manayunk supply, which is more than 100 per cent increase for that section, and up to 200,000,000 gallons will be pumped to the Queen Lane filters to take in that section of Germantown. And the Queen Lane filters have been very recently remodelled by adding what is called a mechanical or "pre-filter" in addition to the slow sand filtration. The adding of the pre-filters in all our filter stations has aided materially in increasing the amount of water filtered. This type of filter is constructed of very coarse sand and stone, and the water is allowed to go through these very hurriedly, taking

out the floating loam, dirt, and stuff of that character which clogs the sand filter, and makes the slow sand filter very costly to operate. Because, when a slow sand filter is put in operation, if it does not work a given length of time, it reduces the capacity of the plant very materially. The filter very rapidly becomes clogged and the rate of the water-flow is retarded, and we are then forced to scrape the mud and dirt off, wash the sand and put it back, and then back-fill the filter and start the water over the sand again. These pre-filters take care of that, as they are cleaned by a process of forcing up under heavy pressure water and air through the stone from the bottom and driving off the sediment to the surface, and then the filter can be put in operation again very quickly.

From the Torresdale filters up to 350,000,000 gallons per day can be filtered, of which 225,000,000 or 250,000,000 gallons will flow through the Torresdale conduit and be pumped into the general distribution from Lardner's Point. The present low-service pumping station at Torresdale will be converted into a high-service system and supply the 45th and 41st wards. At Torresdale, along the river, we have a low-service station which pumps from the Delaware River to a subsiding basin, and from there into the present filters, and then down to Lardner's Point, where it is distributed. So that this station with very little change can be brought up to a high-service station and supply the greatly increasing demand for water in the northeast section of the city.

The pumping stations on the Schuylkill River, at Belmont and Shawmont, will be abandoned. This will not be any loss to the city, because by the time these projects can be carried out the machinery will be of such an age that it will be entirely worn out, and the increased economy brought about by the cross combined turbines at the Queen Lane pumping station will pay for the loss, if there is any, in the machinery to be abandoned.

The filters at Queen Lane, Belmont, Roxborough and Torresdale will be gradually enlarged to meet the demand for water in the respective districts, and this can be done without the necessity

of obtaining more land or the building of filters at other stations. It is proposed to build in the northeast section a supply reservoir, holding about 100,000,000 gallons, for stabilizing the supply of that section of the city.

The plan, as a whole, adapts itself to utilizing at the least expense the existing plants of the Water Bureau. Furthermore, the claims for damages will be relatively small and very much less than any other project, in that the water from both the Delaware and Neshaminy will be taken relatively near to tidewater and consequently the water-rights will be at a minimum. In other words, if you are going to take water from up in the mountains, where it is necessary to protect the stream, you will have to buy thousands of acres of ground to protect the streams flowing down into the watersheds. This is not necessary in this case, because, as stated, it is near tidewater, and the Neshaminy Creek is so logically a reservoir that the ground around there is all that is necessary to be taken, and a right-of-way for our conduit from Yardley, the low-service station thereon down to the Neshaminy.

It will require ten years to build this. I want to be frank about that, because some people talk about the Green Lane plan all being accomplished in four years. I grant they may build a dam there in four years, but, as I stated to you previously, that would be simply a flush for the Schuylkill River, and it does seem to me that that is not what we want, and this project—as I say frankly—the complete project, will take ten years. And that is if the funds are provided as required, and accordingly longer if the appropriations are limited. It is self-evident that the plan must be largely completed before it will be available for use—the Delaware pumping station, the Neshaminy dam and reservoir, the Torresdale high-service station, and the necessary additions to the filters must be built before the new supply can be made available. In other words, we can put the pumping station on the Delaware and bring down the water and use it, but we cannot complete the job in less than ten years. And then, when we are raising the supply to 600,000,000 gallons the addi-

tional filters that must be put in can then be arranged for. The work is so large that it is impossible to build and utilize it in part as a temporary measure.

The loan provides for $6,000,000 for the improvement of the water supply, including the acquisition of the Holmesburg Water Company. Now the department's plan for expending this sum is as follows: In the project of the Green Lane Reservoir it outlines a project of what it will do with the $6,000,000. This is what the Bureau says it will do:

The Holmesburg Water Company, $850,000; the Fairmount dam, $750,000. And I will be almost willing to stop right there and say we do not have to use $750,000 to repair the Fairmount dam or rebuild it, except that until these projects are well developed it will be necessary to have a dam there that will be secure, so as to conserve the water for our pumping stations at Shawmont, Queen Lane and Belmont. And then I suppose, too, it is the city's duty to take care of its property, and under those considerations the money will have to be spent for erecting a new dam, and it comes under the Water Bureau. But I say to you that, with this Delwaware River project, it would not make any difference whether the Fairmount dam went out or not.

Connection between the center of the city and West Philadelphia could be made by a 48-inch pipe across Girard Avenue bridge. This will cost $250,000. That is, to tie in West Philadelphia in case of a breakdown in any one of the West Philadelphia stations, or if for some reason or other they did not function. Thus we have got a tying-in link with the Torresdale or Queen Lane filters and could go on supplying water from that section to West Philadelphia, or we can reverse it, and then—if anything went wrong with Queen Lane—we could supply water to the district which Queen Lane takes care of, through the West Philadelphia filter stations.

Improvement of the distribution system in the northeast: As I said, there is a great need for water in the northeast section. That will cost $1,000,000.

Improvements in pumping stations and filters, $150,000.

Acquisition of property, $1,000,000.

Neshaminy dam, $1,750,000.

Boring, surveying, etc., $250,000. A total of $6,000,000.

You will note that one-half of the $6,000,000 can be used in securing a new source of supply, and that the amount of work that can be accomplished by this sum is not large. It will, however, provide funds to secure the necessary property and will permit the construction of the Neshaminy reservoir to start with, so we will impound the water there promptly.

As I stated in starting out, this plan will cost $60,000,000, and I do not think that it is necessary for an engineer to make any study of a proposition where one gives you 250,000,000 gallons of water per day at a cost of $76,000,000, and the other provides an ample supply of 600,000,000 gallons for $60,000,000.

Any one here tonight having an industrial plant and using boilers, knows that the Delaware River water is infinitely better to use than that of the Schuylkill River, as it is softer, has less sediment and less incrustation takes place in the boilers. Also. those of us in West Philadelphia who have to use Schuylkill water, frequently are obliged to put up with some very bad odors and tastes, especially at this time of the year, due to decayed vegetation along with the trade-waste that lines its banks. I think and feel that the layman should be able to judge what is necessary and what we ought to do.

We talk of a commission being appointed. A commission not long ago furnished a report, in which they took up the question of both the Perkiomen and the Neshaminy, and advised the use of both, providing we had $135,000,000 to spend at once. The bureau chief and the engineers have gone over this very carefully and there is no question in their minds that the safe and sane thing is to take the supply from the Neshaminy Creek, impounding it there, and the balance from the Delaware River, at Yardley, above Trenton. This is a financial problem and one that speaks for itself, and I hope you will urge upon the authorities the very great necessity for this improvement and of starting it as soon as possible. This is not a subject that ought to get into politics. It should be

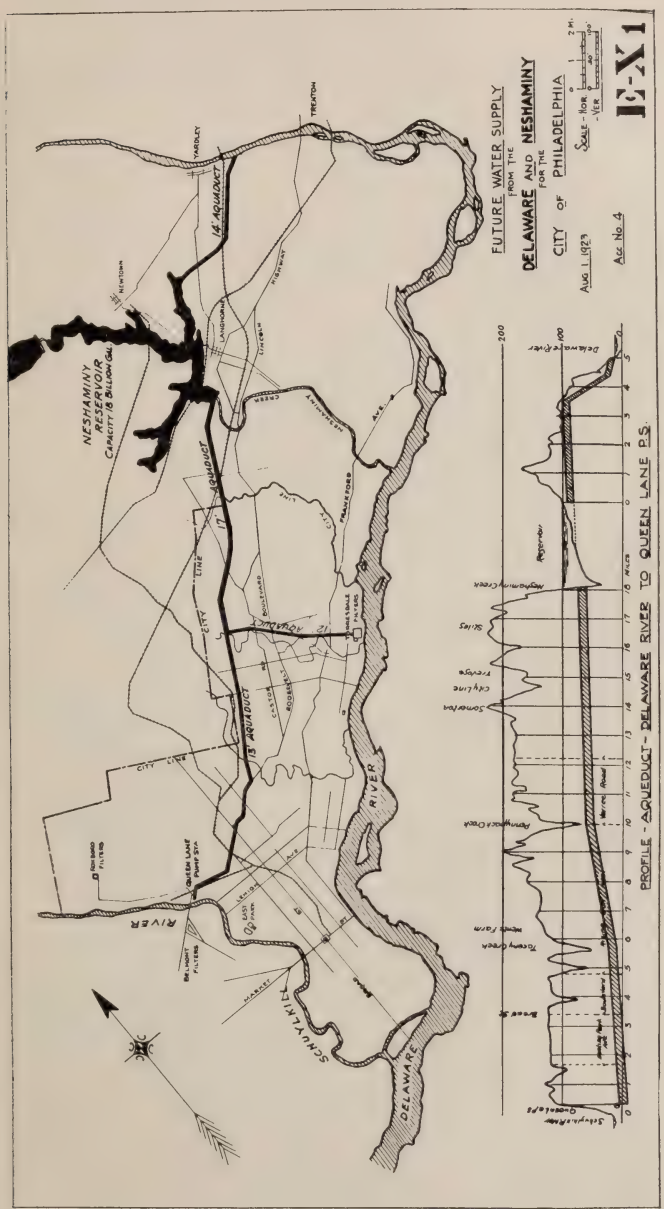
FUTURE WATER SUPPLY
FROM THE
DELAWARE AND NESHAMINY
FOR THE
CITY OF PHILADELPHIA
AUG. 1, 1929
ACC. No. 4
E-X 1
NESHAMINY RESERVOIR
CAPACITY 18 BILLION GAL.
14' AQUADUCT
13' AQUADUCT
12' AQUADUCT
11' AQUADUCT
CITY LINE
RIVER
DELAWARE
SCHUYLKILL
PROFILE - AQUEDUCT - DELAWARE RIVER TO QUEEN LANE P.S.

FIG. 1

FIG. 2.—The Neshaminy and the Tohickon, watersheds in the Green Lane section.

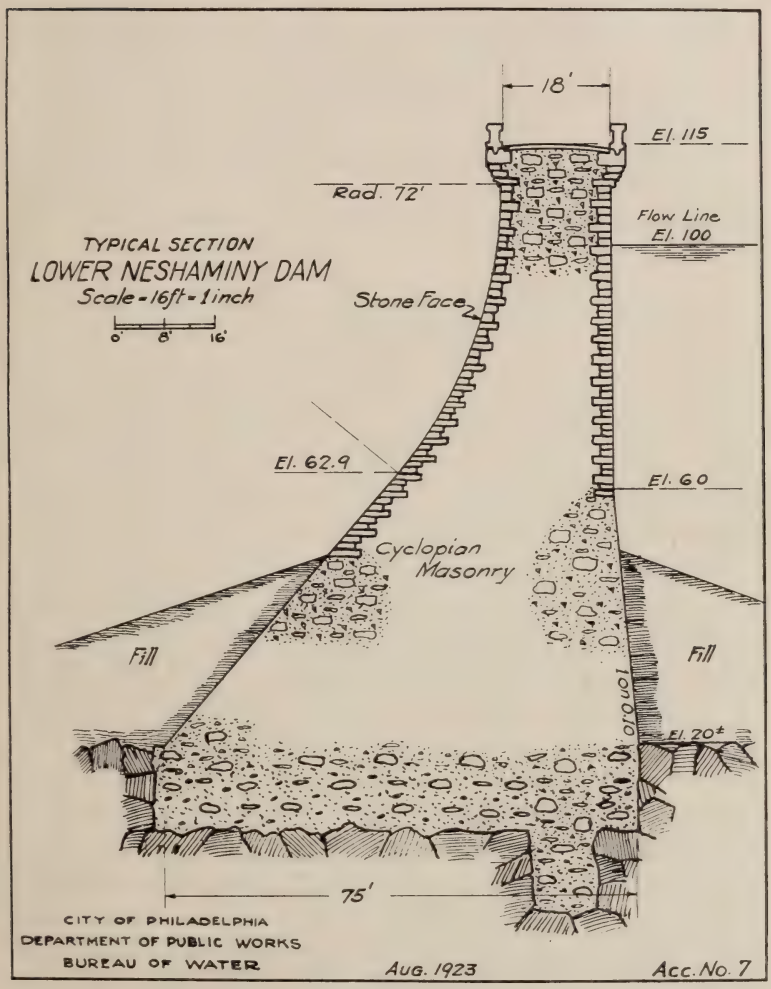

Fig. 3.—Shows a profile of the dam at Neshaminy, showing how deep it will have to go down, etc.

dealt with as I am glad to know such men as Dr. Anders and Dr. Abbott will deal with it, for the health and benefit of the citizens of Philadelphia.

I have a few slides which I can show you, and while they may not be of a great deal of interest, yet will give a general idea of the location of the watersheds of the Neshaminy.

This plan proposes that Philadelphia use the Neshaminy Creek and take the water from the Neshaminy in that watershed and get the balance from the Delaware River at Yardley.

Needs for Modern Methods of Disposal for City's Waste.

There is another subject on which I would like to say a few words tonight, because you are all so vitally interested in the health of Philadelphia; and outside of the water supply, there is nothing that will improve and maintain the health conditions of Philadelphia more than the disposition of refuse, garbage, etc.

I am very sorry that I could not have the plates made in time to better show you exactly what it means, or just what conditions are existing in Philadelphia in many sections, as to the disposal of refuse, etc. I will pass these pictures around and you can see from them just what the city of Philadelphia is suffering from today.

The Bureau is forced to haul away the garbage and refuse from the houses, and carry and deposit it on the dumps (wherever it is possible to get a dump), and there it must stay until there is enough dirt brought to cover it, or it deteriorates and finally reaches the point when it becomes earth. You can imagine that old bed-springs and that sort of refuse do not become earth very rapidly.

In the disposal of rubbish, garbage, refuse, etc., from slum districts, where we get beds and mattresses filled with vermin, they are now being carried away and thrown on the dumps; and this is the method the city of Philadelphia has been using in getting rid of its waste for years. The dumps are breeding places for

vermin, rats, flies, etc., and the garbage in the waste is food for the rats and they thrive and grow like a green bay tree. When a dump catches fire (and it is almost impossible to keep them from getting on fire, owing to the character of the refuse), it is a nuisance not only to the immediate neighborhood, but the odor and smoke are carried for squares.

Not long ago an entire day was spent in south Philadelphia by the fire department putting out fires on the dumps.

Here is a picture of a dump in north Philadelphia, by which you can see a portion is on fire. Here is a picture of the same dump from another angle, also a picture of a dump in south Philadelphia. (Showing photographs.) Fortunately the city of Philadelphia is now taking a step forward. My good friend, Mrs. Oakley, whom I see here tonight, turned the first spade of dirt when the erection of the destructor in northeast Philadelphia was started.

The idea of talking to you just a little on this subject tonight is to have you interest yourselves and urge along the proposition of having more destructors built throughout the city of Philadelphia. Dr. Abbott is very familiar with this subject. He has had such a long and vast experience helping to abate nuisances in this city that I know he thoroughly agrees with me that a refuse dump is a menace to any neighborhood, and we cannot pound too hard at our city legislature to appropriate money for the building of these destructors.

The Bureau was unfortunate in not being able to influence Council to include in the present Loan Bill money for additional destructors. The Loan Bill is made up of a number of projects, one put in to suit this one and another to suit that one, so that all sections represented by these members might be satisfied; and the outcome of it is that there is not one dollar in the Loan Bill for destructors.

Council seems to be afraid to approach the subject of destructors, stating that they do not want one at their door. It is not necessary to locate one at anybody's door. A destructor can be located and built in such a way that it is not a menace nor an

annoyance to any neighborhood. And it seems when you stop to consider that you have three communities—numbers one, two and three—and you have a destructor in Community Number Two, and it takes care of all its own refuse and dirt, that it is hardly fair to haul refuse from Community Number Three through Number Two to a dump in Number One. This is what we are doing every day. You are not only suffering from what is collected in your own immediate neighborhood, but are harassed and annoyed by the teams and wagons hauling refuse not from one community but five communities, and today, for instance, in one section of south Philadelphia where they do not have ashes—just rubbish—every winter they are annoyed by the hauling of the ashes gathered from districts as far north as Poplar Street and Girard Avenue and carried to the Seventeenth Street dump in south Philadelphia traveling through the Girard Estate, the section without ashes. A man in this section said to me that he did not see why his street should be littered up by the ash teams. Unfortunately for him, that was the only route to the dump and there was no other place to dispose of the ashes, etc. That is the injustice of it. The sanitary way is to dispose of the waste by burning it and getting rid of it as near as possible to the community in which it originates.

The destructor used by the city of Toronto has been operating very successfully. It is the largest one in the country at the present time. I talked to a number of the residents in a neighborhood possibly half a block away from the destructor, asking them how they liked having a destructor near them, and they said they had never suffered any annoyance from it. If the people will only stop and consider and help to influence council to appropriate money to erect destructors, it will help the health of the city, abolish a nuisance, and from a financial point of view the city will save money.

DISCUSSION

Dr. Alexander C. Abbott: The matter is now open for discussion, and we will be glad to hear from any one who cares to ask any questions or discuss any phase of the subject.

Mr. Edward B. Chapman: How much was appropriated for that work?

Mr. Caven: All the money we have for that work is the general fund for the building of stables and stations for the Bureau of Street Cleaning, about five hundred thousand dollars. The Bureau contemplated building a destructor at Sixteenth and Cambria Streets, but Council after passing the ordinance repealed it on account of people complaining. They tried several other locations and finally the department was forced to go to Ramona and G Streets and build there. It is an outlying section and it cost considerably more to build the destructor there than it would at Sixteenth and Cambria Streets, as it is on the side of a hill and the foundation ran into considerable money.

Mr. Gaffney, Chairman of the Finance Committee, is commencing to think that south Philadelphia is suffering from these refuse dumps and is willing to get rid of the dumps providing the Bureau can get a destructor located in the section he represents where it will not be an annoyance to any one. That is going to be a hard proposition, because, while it will not annoy anybody, yet at the same time we feel that we ought not to put the refuse from one section into some other section. We feel that the thing to do is to locate the destructors throughout the city in such a way that they will be worth while and will do the work.

I could not help bringing up these things tonight. Dr. Abbott has been so helpful along these lines, as I stated, and my friend, Mrs. Oakley, is also helping it along. We have met so many obstacles in attempting to do this street-cleaning work that we have had to call on the doctors to help us out.

Here is a picture of the old William Penn Hotel, at Thirty-eighth and Market Streets, now turned into one of our street-cleaning stables. Another showing the various stages of the completion of the destructor we are building at Ramona and G Streets.

Here is a picture of a large sewer—in fact, the largest sewer in Philadelphia. Doctor, here is a picture of the last stable you helped the city get. I will be very glad to answer any questions, if I can. I hope you will think something about these subjects and later I am going to ask the doctor to have the physicians take it up in an energetic way. I know Mrs. Oakley has already been doing so, and I am going to try to be in a position to help her along in this work, and see if we cannot bring to a realization some of these things. The city must have these destructors for the benefit of public health.

Dr. Joseph Leidy: Can the state control the situation on the upper Delaware plan, so as to prevent manufacturing industries locating there and contaminating that supply in years to come?

Mr. Caven: Yes. As far as the state of Pennsylvania is concerned, in the last legislature they passed an act—and there is a commission that has charge of such matters—that no industry shall be permitted to build on the banks of a stream if it is going to harm the water in any way.

Dr. Edward J. G. Beardsley: What would be the objection of taking the entire supply of water from the Delaware River, rather than merely as an addition to the water in the dam at Neshaminy?

Mr. Caven: In the first place, to filter water and do it economically you must have a subsiding basin, and you not only utilize the Neshaminy watershed, which brings considerable water in addition to what we would take from the Delaware, but it makes a subsiding basin and the water would lie there many days and subside, so that when it reaches the filters it would be fairly well settled and have little sediment in it, and that would be a great help to the filters.

Dr. Seneca Egbert: Do you think it would be ten years before we got any water from there?

Mr. Caven: No, it would not. The Neshaminy dam would be built at once, and we would avail ourselves of all the water in the Neshaminy watershed.

Dr. Egbert: Well, how long would it take to complete the aqueduct so the water could be delivered to either one of the filter stations?

Mr. Caven: The aqueduct from Neshaminy to the filters would take four or five years.

Dr. Egbert: That would be finished very shortly after the completion of the dam?

Mr. Caven: Yes, very shortly after the completion of the dam. If the money were available, work could start now, as far as the aqueduct and the dam are concerned.

Mrs. Oakley: Mr. Caven has spoken about the destructors, but he hasn't mentioned anything about the by-products. I do not know whether we are to get anything out of ours or not.

Mr. Caven: No, there will be no by-products.

Mrs. Oakley: Well, if Mr. Caven could retain his directorship he could help that destructor plan along very much.

I have made a considerable study of destructors in London and Paris. In London all garbage, rubbish and waste go into the destructor, which is not very far out from London and is not at all a nuisance. Electric power and light are obtained from the furnace. The waste burns without coal and gives numerous by-products. In London, the ash which comes out is mixed with cement, also the ground-up fused glass and china, and from that they make bricks which are used for paving the streets, building fire-backs and furnaces and other things. (Shows sample of brick.) They are practically waterproof and fireproof and are also practically indestructible.

In a small town near London they have a destructor in which they burn all refuse and get enough power from it to pump the water into the reservoir. They have five tons of refuse a day, and five tons equal one ton of coal, and as I said it makes enough power to fill the reservoir every day. Another town, also near London, uses the electricity from the destructor to run a local electric railway.

Now in Paris they have almost a super-destructor. In the first place, they collect it in a "super" way. When I first arrived in Paris I wondered what they did with the garbage cans, for there were practically no alleyways. I found out later that they let each block know exactly what time the garbage cart would arrive, and the people put out their garbage cans ten minutes before, and the cans must be taken in ten minutes after the cart has left that square. In the square where I lived the garbage cart arrived at 7 A.M., and ten minutes before seven everybody would be putting out garbage cans, and when the garbage cart arrived and passed down the street it was a very remarkable sight. It was a long, low, tubular cart, like one of the Standard Oil trucks, and the lid was in three divisions. When the first division was filled that lid was clamped down; then the others in turn, and when the cart is full it is taken to the destructor. The cart is quite low, so that when a man wants to empty garbage into it he does not have to do it as they do with our garbage wagons. He empties quite naturally at his own level. Then this cart goes, as I said, to the destructor. I followed one of them, and the Director of Public Works of Paris and I went through the building and were shown everything to be seen there. It was a most amazing plant and would not have been a nuisance in the very best part of the city. It was in the form of a large square, and on one side the men who attended to it left their clothes in lockers, and each one had a shower bath, so after his day's work he could go home clean. On the other side was the furnace, and on the third side a long pit—sort of a tunnel—and when these cars came in and were tilted

up, and the lower part of the lid taken off, all the refuse was dumped down onto a moving platform. Beside this platform sat two men with rakes. They watched the refuse as it came out and retrieved anything that seemed to be valuable or uninjured—old dishes, for example, or a whole pair of shoes or something of that kind. Not very much, of course, goes to waste in Paris. Then the waste was taken to the destructor and burned, and the product was electric lighting for the city.

I think there are three destructors in Paris in different parts of the city. In London they have only one, because lying on the river they let much waste go down to the sea rather than haul it.

In New York, in the ashes dumped into the sea, 150,000 tons of coal, anthracite, are wasted annually and could be used to advantage somewhere else. They burn it in destructors in England, and, as I said, it helps to pump the water. I think the only successful destructor in our country is in Milwaukee. It is the only one that utilizes by-products. In America we do not put electricity to work because there is always some U. G. I. or electric company that has a perpetual franchise, but in Milwaukee the electric power generated in the destructor is sold to private business.

Mr. Caven: I might have said, in talking about destructors, that Philadelphia is depending for the removal or the destroying of its garbage on the plant at Forty-ninth and Schuylkill Avenue, and all its eggs are in one basket. Under contractor government, the last year, the peak of the contractors' work was 51,000 tons of garbage. Last year—the city of Philadelphia doing the work—we collected 106,000 tons of garbage, and this year we will go beyond that. Council appropriated $155,000 this year for the operation of the garbage-reduction plant. That was to run it for six months. The first day of July the Bureau of Highways turned back to the city of Philadelphia $205,000 less $17,000 for depreciation. In other words, we gave them back every dollar they gave us for the operation of the plant and $52,000 profit. And I only cite that so you can see what can be done and it is not hard to do if you are playing with the cards on the table.

Dr. Egbert: It seems to me the interest of the general public should be awakened as to the importance of this movement for an increased water-supply, especially in West Philadelphia. I am one of the unfortunates who live in West Philadelphia and have to take the taste with the water. It is not a question of politics—it is a question of quantity and quality, and as the city is growing so rapidly—from the diagram shown here—we ought to realize what we have cannot be sufficient much longer and we must have another supply. Now there is, as you

know, a controversy as to which is the best plan. I think the Director made it clear to everyone here who did not know it before, that there is only one answer to the question, namely, that the Neshaminy-Delaware supply is the safest, wisest and cheapest way for us, and it seems to me that everyone that goes out of this room tonight should try to get people wakened up. I know you, Mr. Chairman, and your clerk have tried to get a big audience here. This room should have been packed. Many times more than enough people to fill this room were invited here tonight but are not here, and so it seems to me that the people in West Philadelphia especially should be awakened to the importance of coming to a right decision, and working for this new and big water supply.

The reason why I asked whether we should have to wait ten years, was simply to bring out that we could and would get water in much less time than that; for if we told the people the project would not be completed for ten years, it would, of course, have a disheartening effect on them. Many of them feel it would be a waste of money and time if it would take that long. But if they could get an abundant supply of good water in a comparatively short time, it would help us to put this over. I believe that every man who goes out of here should go with the full determination to boost the project and stand by the Director's plan as against any other.

Dr. Randle C. Rosenberg: I am glad to hear that the Perkiomen is not going to be used unless through an aqueduct. I am a summer resident along the Perkiomen and can see what happens there. It has no chance to purify itself, say, as the Schuylkill, and I am awfully glad to hear this.

Dr. Abbott: In connection with what Dr. Rosenberg has said, I will ask the Director if the figures given by him for the cost of the Perkiomen supply include the aqueduct.

Mr. Caven: Yes, that included the aqueduct.

Dr. Leidy: This is a perennial subject for discussion. It comes up every two or three years—the water-supply of Philadelphia. Just one hundred years ago the population of the city of Philadelphia proper was 50,000. Including the outlying districts it was just 50,000 more. We know what the population is today. I should like to ask the Director, has any consideration been taken or made note of in connection with the continuous and increasing contamination of the Delaware River? That is a grave proposition. How far is this Neshaminy proposition going to relieve us from the standpoint of the future, with the increase of population and the increase of these manufacturing plants? Most

of us here remember as children the cleanliness of the Delaware River. We know now from the report of the Fish Commission what that condition is. It is practically destructive to all forms of animal life. I should like to know how far we will be able to go to stop that.

Mr. Caven: That has been under consideration. The Delaware valley above Yardley will never be populated to the extent that the valley of the Schuylkill is. It is chiefly a farming district. The Schuylkill valley is a mining and a manufacturing district. Now the Delaware has got such a large flow that the contamination would be very small, particularly since the states have joined together and are now exacting of each municipality or town to put in sewage-disposal plants, and, as I stated, they passed an act at Harrisburg, that now no one can erect a plant and discharge wastes into the river or anything that becomes a nuisance or an injustice or injury to the water-supply of any borough, township, etc.

Just along that line, I might say that in building what we call the Southwest Sewage Disposal Plant in Philadelphia we were anxious to put an end to the evil effects of using the Schuylkill River below the dam as an open sewer. The sewage will be conveyed by mains to the Southwest plant and there pumped out of the sewers into the septic beds, treated chemically and then discharged into the Schuylkill River. When I asked the State authorities whether they would permit us to pump the sewage directly into the Delaware without first treating it on the septic beds, they said, "No." We said, "Well it is going into the Schuylkill River now." They said, "Yes, but you are much too near Chester, and we are not going to permit you to put your sewage so much nearer Chester. It is bad enough to have it come out of the Schuylkill, but we will not let you put it in the Delaware because it might affect the water at Chester." So you can see how they are guarding one town from the other. Philadelphia is very fortunate in having the Schuylkill, because it is a sort of a sedimentation basin. The tide holds the sewage and it has a chance for settling, and by the time it goes into the Delaware it has had some treatment. I just cite that to show you that that is the idea.

Now Jersey—they have gone up to Trenton, one of the great offenders—has joined with Pennsylvania; they are putting in their sewage-disposal plant. That is a very nice subject, because we as citizens of Philadelphia in the future will have to meet an expense, in the disposition of our own sewage equal to the cost of operating our water works today, and the expenditures for the erection of our sewage-disposal plant will cost

$53,000,000 or more. We have to handle thousands of tons of sludge each day and it costs as much to operate the sewage-disposal plant as it does to furnish drinking water to the city.

Dr. James M. Anders: The presentation made by Director Caven is both timely and important. He reviewed the detailed facts of the two main propositions which have been advanced with a view to obtaining for Philadelphia a better and more ample water supply. There can be no room for doubting that the reasons adduced by Director Caven in favor of the plan which advocates going to the upper Delaware and the Neshaminy for the new water-supply are all sound. Indeed, it is high time that the Schuylkill River be abandoned as a source of our water-supply. I agree with Director Caven that there is no likelihood that as many manufacturing plants will line the banks of the upper Delaware as are to be found along the Schuylkill River. Moreover, the plan advocated by Director Caven will meet the city's needs for the ensuing fifty years.

Surely, the present-day so-called "dumps" for the deposition of rubbish, garbage, refuse, etc., are insanitary. The Director has not overdrawn the picture. The need of destructors in different sections of the city is obvious. At present much of the city's waste is hauled long distances, the wagons scattering fragments as they go on their way to a dump, which is thus made a growing menace. On the other hand, sectional destruction plants are not a menace, if properly located. They should, of course, be erected only in business or industrial districts. I can corroborate what Mrs. Oakley has said about the superior methods of collecting garbage, refuse and the like in Paris and Berlin, by comparison with our own. I do not take the same gloomy view that was expressed by Mrs. Oakley with regard to the possibility of having the efforts so well begun by Director Caven continued by others during the next administration. If those interested in seeing sectional destructors established unite their influence, and keep up agitation of the question, they will probably bring about more or less satisfactory results.

Dr. Abbott: And now you see how interlocking all these activities are. Mr. Caven has told us of some of the obstacles. Nobody wants the destructors near them. Suppose the city were zoned properly; there would be distinct and definite industrial neighborhoods. There could be no legitimate obstacle to locating incinerators in such districts. At the present time nobody knows where they might be put.

I shall not forget an impression that I got the first year that I was in

office. A very decent-looking woman called and said she wanted to complain because somebody was throwing garbage out on the lot in front of where she lived. I went myself to see what the complaint was. She gave her address as somewhere on Snyder Avenue; when I got there I was astonished at what I saw. Looking south, it was like viewing the desert of Sahara. In every direction was rubbish and some sort of refuse and vehicles dumping more. I suppose they are still dumping it there. It was the most extraordinary thing I ever saw near civilized human habitations. Somebody remarked, "That ground has got to be filled up." I suppose it has, but I do not see why it should be done with such materials.

DECEMBER 10, 1923

The Relation of the Oral Hygienist to Public Preventive Medicine

BY W. A. JAQUETTE, D.D.S.

Years ago certain members of the dental profession realized that the filling of teeth and crowning those whose destruction had made them past filling was not enough. Such treatment did not consist of sufficient care to ensure the preservation of those organs. For even the best of such care could not be always counted on to permanently save the teeth. Cleaning the teeth with a toothbrush has for years been recommended and urged by most dentists, but it still seemed that conditions did not very materially improve. Dr. D. D. Smith, of Philadelphia, was one of the first dentists to systematically take up the work of oral prophylaxis. This consisted not only of the removal by instruments of all deposits of calculus, but the removal as well of the mucoid films by which the majority of stains are glued to the surface of the enamel, and in addition the massage of the gums.

"The various forms of mucinous and bacterial plaques which cover all neglected and unclean surfaces of the teeth are of as great importance as, if not greater than, the dental calculi. It is in these films that the salivary calculi and stains first become impregnated

and are attached to the surface of the tooth. So also do they afford an excellent medium for the propagation of oral micro-organisms, many of which when growing in this manner exert a harmful influence upon the teeth and surrounding tissues. For instance, acid-producing bacteria beneath a plaque may produce sufficient acid to decalcify the enamel and form an initial lesion of caries. Other types of organisms when growing in a plaque contiguous to the gums at the neck of the tooth frequently exert a toxic and irritative effect upon these tissues, producing chronic gingivitis and a tendency toward pyorrhea.[1]"

D. D. Smith, by his skilful prophylactic treatments at stated intervals, accomplished such gains in mouth hygiene that dentists came from other cities to witness. Among those visitors was A. C. Fones, of Bridgeport, Conn., who conceived the idea that young women could be trained to do much of this work by providing for them a course of instruction much less extensive than that for dentists. He established a school in the fall of 1913 and secured for the faculty nineteen men of prominence, enlisted largely from the eastern universities. Among the subjects presented were anatomy, physiology, bacteriology and sterilization, deposits and accretions upon the teeth, chemistry of food and nutrition and personal and general hygiene. In addition to the lectures, clinical instruction was given to the class of 32 students, who were all graduated as *dental hygienists*.

So a new profession was established.

Some of these dental hygienists went into private practice and into hospitals, but many went into the schools, where their service is most effective.

In the Bridgeport public schools operating-rooms were improvised on stair landings, basement rooms and wherever space and light could be had. Appropriations by the Board of Education were increased so that it was possible to give each child in the schools of that city from one to three prophylactic treatments during the school year. At the Health Center, provision was made for extractions and fillings at a very small sum or even without

cost to those who were unable to pay. A survey of the Board of Education of Bridgeport in 1912 and again in 1918 showed a remarkable change in the rate of *retardation*. These five years paralleled the activities of the health center in connection with the work of the hygienists in the schools. The average drop in retardation for the first eight grades was 50 per cent. The cost of reëducation in Bridgeport for 1912 equalled 42 per cent of the entire budget and for 1918 equalled 17 per cent of the entire budget. The demonstration of such financial saving made possible the securing of funds to carry on the work. Toothache has been found by school nurses and dental hygienists to be a frequent cause of absence from and poor work in school. Dental abscesses to even a greater extent are responsible for such delinquencies. The improvement in health and in school work in Bridgeport has been such that the Board of Education has passed a ruling that no child may be promoted from the fifth to the sixth grade who has decayed or unclean teeth.

Mouth hygiene plays an important part in communicable diseases which involve the respiratory tract through the mucous membrane lining the mouth, throat and nose. A neglected mouth which contains decayed teeth, decomposing food, red and congested bleeding gums presents not only the incubator but the pabulum as well for organisms, and it is in this further connnection that the dental hygienist is valuable in disease prevention during the school period when contagious diseases are especially prevalent.

The licensing of dental hygienists by the different states has met with some opposition, but the number is increasing, until now we have twenty states that have passed laws outlining the work of this new professional group. Dental nurses they are called in some states. In Pennsylvania the law requires that the applicant for a state license shall have had a full four years' high school course, a year's work at a recognized school for the training of dental hygienists and a year's internship in a school, hospital or sanitarium. Though the law in this state was passed so recently, and but two classes have been graduated in our training schools,

there are at the present time 55 dental hygienists in *public* health work in 32 municipalities or centers, financed as follows by:

School boards	28
Hospitals	2
County and local tuberculosis societies	7
Red Cross	4
Child federation and kindred associations	4
Industrial plant	1
State sanatoria	9
	55

The important facts of oral health the dental hygienist teaches to the school children in classes, and individually when she takes each child in her operating-chair for mouth prophylaxis. This *specialist* in mouth hygiene is able to enlist a greater child interest and coöperation than the physician, the dentist, the parent, the nurse or the teacher. Good hygienic habits formed during the plastic period of childhood become part of the make-up of the individual, and if adolescence is passed with good dental organs, their preservation throughout life is likely assured. All children in the schools, the great national asset, should have this protection.

I would here most respectfully submit that the dental hygienist is not a new type of manicure artist—a product of the beauty parlor—but a most efficient instrument in public preventive medicine. Her work in hospitals and sanatoria already has been to contribute to improved health conditions.

Mouth hygiene is influenced by (1) nutrition (prenatal and during the nursing period especially), (2) functional stimulus, and (3) sanitation.

1. Nutrition of the prenatal and nursing periods is so critically important, because the first permanent teeth are beginning to calcify prior to birth and soon after. Calcified tooth enamel, unlike most other tissues of the body, is without circulation. Therefore the quality and arrangement of the enamel rods laid down remains unchanged. Nutrition is the field of the physician,

but I should like to emphasize the fact that dental conditions are dependent fundamentally upon proper feeding of mother and baby.

2. The importance of functional activity is not appreciated by the laity nor indeed by the members of the dental profession. Chewing is natural to the animal, as shown by the young dog with an old shoe or a young child biting his toys, etc. The chewing of food not only prepares it for digestion but stimulates the circulation of the blood within the pulp of the tooth as well as the supporting tissues. The result of good, vigorous chewing is to create more massive alveolar processes and fibrous tissue mat—the gum tissue. The teeth are therefore the better able to withstand occlusal impact and the gums to resist trauma in chewing and bacterial invasion. There is also developed wider arches with consequently room for the permanent teeth to erupt in regular formation, and the proper function of the whole dental mechanism is kept intact, and probably better air passages are developed as well.

The third factor influencing mouth hygiene is sanitation. By the removal of all hard and soft deposits and stains with food debris, putrefaction in the mouth and alimentary tract is lessened; much of the fermentables that cause decalcification of the enamel surface are gotten rid of, and by the removal of the bacterial plaque contiguous to the gums those tissues are freed from that tendency toward gingivitis and pyorrhea which is due to the toxic and irritative effect of the organisms. After the hygienic state of the mouth is restored this condition must be maintained by the intelligent and interested coöperation of the patient, who must be carefully instructed. The work of the dental hygienist is most important in teaching and maintaining oral health, but the responsibility for knowledge of pathological conditions rests with the medical and dental profession.

Dental caries presents one of the most important of present-day public health problems. It is active in 95 per cent of our school children. The primary result of dental caries is the retention, in the cavities of decay, of remnants of food undergoing fermentation and putrefaction. Through such a mouth, food, no matter how

carefully pasteurized or sterilized, enters the stomach polluted. The subsequent result of dental caries is the exposure of the dental pulp. This tissue, the pulp, may be considered the formative organ of the dentine and is made up of an arterial and venous system, a nerve supply, odontoblasts and connective tissue. With the exposure of the dental pulp there is infection of that tissue ending in necrosis. As the root canal has one or more openings at the root end, microörganisms of the mouth may be most readily implanted in the deeper layer within the cancellous tissue of the bone.

An editorial in *Hygeia* for December, 1923, quotes the Department of Health, Newark, N. J., as recently formulating ten brief statements in reference to heart lesions. Following are three of these statements:

"1. Heart disease is nearly always the result of rheumatic attacks in childhood, either acute or chronic.

"2. Rheumatism is an infectious disease caused by bacteria, and is most prevalent in spring and fall months.

"3. Permanently enlarged tonsils and adenoids are the gateways through which the rheumatic virus gains entrance."

No mention is here made of the large number of carious teeth which are similar gateways through which infection enters.

From the report of the Chief Statistician for Vital Statistics, Department of Commerce, for 1921 one in every five deaths was due to diseases of the heart or nephritis. Since pathologists and clinicians are more and more coming to believe these two diseased conditions are in many instances traceable to focal infection, the demand for mouth hygiene is the more apparent.

To present the microscopic picture of the periapical and gingival lesions of the mouth in the most accurate manner, I have consulted J. L. Appleton, professor of microbiology and bacterial pathology, Thomas W. Evans Institute, Dental School, University of Pennsylvania, and he had kindly prepared for this paper the following statement:

"The usual types of organisms, regarded as of clinical significance, found periapically and gingivally, are streptococci, pneumo-

cocci and the pyogenic staphylococci. The last group is, as a rule, surprisingly poorly represented either in 'health' or disease. The pneumococci, found in the mouth except in individuals suffering from pneumococcal pneumonia, usually belong to the group designated as 'Type IV.' Postoperative pneumonias caused by representatives of this type may be regarded as of oral origin. In individuals suffering from pneumococcal pneumonia, the type found in the mouth usually belongs to the type responsible for the pulmonary infection. The usual oral streptococcus is one of Theobald Smith's and Brown's 'alpha type,' otherwise more generally known as S. viridans. Hemolytic and indifferent types are found often enough, however, to occasion no surprise. Micrococcus catarrhalis has been described as being particularly abundant 'normally' in the gingival trough of children.

"Anaërobic bacteria are known to occur periapically. Spirochetes: Noguchi's Treponema microdentium, T. macrodentium and T. mucosum have been described in 'pyorrhea' pockets; Vincent's spirochete and the fusiform bacillus may be observed not only in surface infections but also in periodontoclastic processes or even in the clinically 'normal' mouth. A word about the pathogenic significance of these organisms: Periapically, present attention rightly or wrongly is limited to the streptococci. I believe that the periapical lesion is infectious in origin and nature; also that it may act as a focus from which are disseminated poisons or microörganisms or both, initiating under appropriate (but unknown) conditions, secondary lesions, *e. g.*, arthritis, endocarditis, etc. The infectious factor, I believe, in periodontoclasia—pyorrhea—to be secondary. This does not mean that it is unimportant when once operative, and it invariably becomes operative early in the disease. It complicates locally the condition and cannot be ignored. It can also act as a focus. It can contribute to intestinal infection, especially in partial or complete achlorhydria.

"The course of pulmonary tuberculosis is unfavorably influenced by neglect of oral hygiene. The lung tissue affected by tuberculosis with its resistance lowered is the more readily infected by

the mouth organisms — streptococci, staphylococci, and other microörganisms with resultant cavity formation. Neglect of oral hygiene in individuals suffering from pulmonary tuberculosis predisposes to secondary oral tubercular localizations.[3]"

There is *another* type of pathological though nonpathogenic disturbance which a broad oral hygiene program must take cognizance of, *e. g.*, those nervous reflex cases which are due to crowding of teeth in the arches or impactions of unerupted teeth producing irritation of the nerve-endings.

To illustrate the type just mentioned, I should like to present 2 case histories of patients of Dr. John V. Mershon, whose work has to do with correcting malformed arches and irregular teeth.

Case I.—Female, aged fourteen years.

Chorea for eighteen months. Very poor appetite; did not sleep well; was at the bottom of her class in school. Her upper right and left second decidous molars were wedged between the first bicuspids and six-year molars, and were apparently being driven farther up into the process. These two deciduous molars were removed July 1. The following October the mother reported that her daughter had had no attacks dating from the removal of the teeth; all symptoms seemed to disappear. Her appetite was good; she slept well and in her midyear examinations she jumped from the bottom to the top of her class. That was eight years ago, and there was no return of the symptoms and she is developing well. (This case I have had an opportunity to observe and can corroborate the above statement.)

Case II.—Male, aged nineteen years.

An unerupted right cuspid tooth with insufficient room for it to erupt. The boy was very erratic and unstable; for instance, he would start to school and would not get there. The unerupted tooth was very deep in the process and was surgically uncovered. There seemed to be immediate relief and a change in the boy's mental condition. Orthodontic appliances were placed and an undue amount of pressure applied to that region. The next day

the old symptoms reappeared. Easing up on the pressure seemed to produce a change toward the normal. The tooth has been brought down into place and there has been no reoccurrence of the symptoms. That was seven years ago.

Both physicians and dentist are prescribing for general nutrition. With special thought as to the formation and preservation of the teeth a plea is made for an understanding of the particular requirements. Aside from the protein, carbohydrate and fat content, with the vitamines, the *mineral elements* are of such great importance in tooth-building and are so often deficient in the American diet, of muscle meat, white bread and peeled potatoes, all of which are demineralized foods.

The dental profession asks of the medical profession that foods be recommended in a state that requires vigorous chewing so that functional activity may produce well-developed and well-nourished dental tissues, teeth and gingivæ. Incidentally, teeth used most vigorously are freest from bacterial plaques which precede superficial decalcification. The dental profession further asks that the fermentables, starches and sugars, be kept to a minimum for the resultant lactic acid precipitates the mucin of the saliva, emeshing the organisms of the mouth and also becoming a bacterial plaque.

If the pathogenicity of streptococci, pneumococci and pyogenic staphylococci be accepted, and these organisms are demonstrated to have penetrated *via* dental caries and the gingival crevice in a pyorrhea ulcer, then *all* agencies for the promotion of *oral* hygiene must continue to be closely associated in the problem of general hygiene.

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DISCUSSION

Dr. Charles R. Turner: I think Dr. Jacquette's presentation of the historical development of the oral hygiene movement and of the

dental hygienist is sufficiently adequate to require no further comment and his presentation of the general problem to which this young person addresses herself and the relationship of oral hygiene to public health seems to be amply and completely covered. I shall, therefore, in the few remarks which I propose to make, confine myself to two closely related matters, which might be called ancillary to the main subject.

I suppose it is perfectly clear to all of us that preventive dentistry must necessarily concern itself almost wholly with children. The problems of adult dental service and child dental service are quite different. When we realize the simple economic fact that, by very careful estimate, there are in the country only about one-third the total number of dentists needed to attend to the purely reparative work at present required, it is evident that the prevention of dental disorders must be considered as perhaps a work of even greater importance. The dental hygienist has come into existence as the expression of another effort to meet the dental needs of the people of this country.

Now, preventive dentistry, which should begin with children, should include these various things: First, the periodic cleansing of the teeth by the hygienist or by the dentist, and the daily cleansing which the individual can perform at home. Second, instruction of the child in mouth hygiene and dental hygiene. Third, instruction of the child and probably the parent as to diet. Whether this should be included within the function of the dental hygienist eventually or not I am not prepared to say, but there is certainly need for further instruction of the public in these matters at present. The dental hygienist can serve a very useful purpose in imparting such general principles as are at present known. Fourth, instruction of the child in general hygiene. This, also, may be an infringement upon the functions of the public-school nurse, which, may I say emphatically, no one sincerely interested in the movement desires, as the public-school nurse should be more or less supreme in her field. May I venture to suggest, however, that the public-school nurse should also receive more adequate training in oral hygiene, so that where the dental hygienist is not available she may serve a very useful function in this phase. The functions of the dental hygienist should be to give prophylactic treatment to the teeth and mouth and to teach oral hygiene; these, in brief, are her two functions. Now that they are of perhaps equal importance, one would be insufficient and incomplete without the other. Dr. Jacquette has brought out, I think very clearly, the importance of a thorough cleansing by one skilled to do it, and with the proper appliances thorough cleansing of the teeth for the removal of mucoid films and accretions and deposits which are not only usually inaccessible to the toothbrush, but are irremovable by the toothbrush, however vigorously applied. It has

been estimated by those who have had experience in dealing with this matter—Dr. Cross, of the Forsyth Institute, Dr. Burkhard, of the Eastman Dispensary, Rochester, and others—that one, two, three cleansings should be performed for each child. They infinitely prefer one thorough cleansing, to be followed up, of course, by a complete regimen of home care on the part of the patient.

Now we may naturally ask, or we should ask ourselves, what are the public or private instrumentalities through which the dental hygienist may be called upon to functionate, just as we may ask ourselves how some sort of reparative dental service may be instituted for those children because 95 per cent of them—as Dr. Jacquette, I think, quoted—are in need of reparative work. The consensus of opinion is that if one had to choose between these two, if we had to face the alternative of having one or the other, it would be better to have the purely prophylactic work, but we realize the urgency of the need of the reparative service. So that while we should aspire to provide the maximum of service of the latter, we cannot hope to provide that in anything like adequate quantity at the present time or indeed probably for a long time to come. We have, however, in this country a number of dental clinics in which the various reparative operations for children and for adults may be performed. Our country is not the most advanced in the development of the school clinics. They originated in the city of Strassburg largely through the activities of Prof. Jessen, of the University of Strassburg. They were quite widely distributed in Germany before the war. In England, backward as England is in matters dental in general, there are about 350 school clinics with about 300 dentists, about one-quarter of whom are full-time dentists. The most advanced program of both preventive and reparative work in dentistry, as far as school children are concerned, exists in New Zealand, where dental clinics are established in many of the schools, sometimes in buildings adjacent, and where all children are provided at government expense with the reparative measures and the preventive measures deemed necessary.

In this country our dental clinics are distributed in the following ways: We have, first, clinics in dental schools. Let me emphasize the fact that these are not primarily the most suitable places for reparative or preventive work for children unless there is an oral hygiene clinic there also. The needs of dental practice, which must be served in the training of dental students, require the establishment of a clinic in which reparative dentistry is chiefly done and where what might perhaps be called the higher reaches of dental work may be learned. Preventive dentistry as a principle for propagation among patients is taught to dental students, but the dental clinic itself is not the most auspicious place for the treatment of school

children. Secondly, hospitals and dispensaries quite often have both hospital and out-patient dental dispensaries. There are not as many as there should be in this country. In 1922, in a survey made by Mr. Michael Davis, of the 144 hospitals that replied to his letter, only about one-half of them had dental dispensaries. We have no accurate figures, but it is quite certain that there are not enough. Now, these dispensaries would serve not only a useful purpose in providing the means for a dental examination of the patients before they are admitted, but if a sufficiently equipped and manned out-patient department existed, it would undoubtedly be convenient for patients who come to the hospital for other purposes to have the simpler and more readily performed reparative work done. Thirdly, we have dental clinics in connection with commercial industrial establishments. In 1922 there were 72 of these known to be in existence in the United States. These, of course, are conducted by the companies or corporations for the benefit of their employees. These are, of course, chiefly reparative clinics. Fourthly, we have dental clinics which are in existence in connection with public health centers, or as public-health centers, of which there may be several divisions in the public schools, or private schools, or parochial schools; and independent or local clinics, many of which are organized through some patriotic or interested group of persons. Many of them have been conducted by dentists themselves on a gratuitous basis, many are supported by Red Cross and other activities, and, also, under this grouping we have a few instances of the dental dispensary as a part of a community health program. Now there are two oustanding examples of this in the Forsyth Dental Infirmary in Boston and the Rochester Dental Dispensary in Rochester, New York. It was hoped that a third one would be founded in Chicago through the generosity of Mr. Julius Rosenwald, but I believe it has not so far materialized. Mr. Rosenwald had a very careful survey made of this whole question throughout the United States, the results of which were published in a brochure entitled *Community Dental Service*, which contained the most recent collection of data relating to this matter, and which anyone interested would, I think, find it extremely useful to read. We hope, certainly, that through some of these agencies the necessary reparative dental service for children may be instituted in connection with and in supplement to the general dental health program which is being carried on throughout this state, as has been referred to by Dr. Jacquette.

The second and final aspect of Dr. Jacquette's presentation to which I wish to allude, has to do with a question which it is a little embarrassing for me to discuss, that is, the question of money. I refer to the fact that this investigation, made by Mr. Davis, included, among other things, a

recommendation that it was a proper and necessary policy for the dentists who serve in these dental clinics to be paid for their services. Now I am perfectly well aware that, as a bald statement, that sounds very unprofessional, and as a bald statement, it is very unprofessional. Let me put a little patch of hirsute adornment over it if I may and soften the asperities of it. The practitioner of medicine and surgery has for years performed an extensive gratuitous service in connection with hospitals and dispensaries, and this has been in part no doubt due to the generosity of the physician in relation to the duty which he felt he owed to humanity; but there is also the fact to be considered, which the physician will gladly admit, that this service also brings in a return in the wide experience which he gains by contact with the great variety of cases which is of very great interest to and value to him. Now the dentist, on the other hand, who works in the public clinic for a few weeks, has covered the whole range of experience which he is likely to have there. He may occasionally get an interesting case, and as time goes on and the importance of the interrelation of dental disorders to general disorders becomes more and more felt, and the consultations which occur between the dentists and. the other members of the medical and surgical staff become more frequent, he will undoubtedly get a very useful return in the experience which he acquires. But dental work is of a peculiar character in relation to the time element. Time has been said to be the dentist's stock in trade. I do not exactly like that phrase, but it allows me to introduce the point that it does require the expenditure of a great deal of time to perform the mechanical work upon the patients, and so Mr. Davis has concluded for these and a number of other reasons which he cited, that at the present time it is impossible to obtain dental service for public clinics without salary. I think this is a justifiable charge to the agencies who are promoting this particular service. Whether or not this should be true of the hygienist I am not exactly prepared to say. The law in this state requires that the dental hygienist must serve a certain period of time before she is entitled to register as a dental hygienist. The primary purpose of that was to provide the state of Pennsylvania with a group of trained women who would be available for this service and before they were attracted to the emoluments of private practice. The law was originally framed for this purpose. The supply of hygienists has rather outrun the demand. The opportunities for service in various school clinics, etc., have not yet reached the point where there is an overplus of demand, so that I do not know exactly what the future of that situation is to be. In concluding my remarks, may I compliment Dr. Jacquette on his presentation and say that I have listened to it with a great deal of pleasure?

Dr. James M. Anders: I cannot let such an admirable presentation as Dr. Jacquette has made and the illuminating discussion by Dr. Turner pass without comment. I would like to congratulate Dr. Jacquette on his interesting story of the movement in mouth hygiene which he represents and the satisfactory way in which that movement has been progressing under his leadership. I am one of those who believe the mouth is a very important portal of entry to the system for disease microörganisms. Therefore, it follows that if the mouth be kept in perfect hygienic condition disease and suffering must in consequence be greatly diminished. I was wondering, while listening to the paper, just what percentage of the public and particularly of the children are reached by the oral hygienists. Perhaps that is not as yet known. From present indications and reflection on the agencies now at work, one has a right to suppose that a much larger percentage of the population will give proper attention to mouth hygiene in the immediate future than in the past, which is an encouraging and significant sign of the times. I recall a conversation that I had with a leading dentist, not long since; he told me that he had been to a social meeting of a few leading dentists of the city only a few years before, and they naturally "talked shop," and the question arose as to what percentage of the population was brought in contact with the dental profession, and one man declared that it was not over 5 per cent, and adhered to that statement. Surely, the percentage is much larger at the present day. Dr. Turner has pointed out the fact that there are too few dentists. I have thought that for some time; dental schools are not graduating sufficient dentists to meet the demand for reparative work. Surely, if the movement represented by Dr. Jacquette's work and leadership develops properly the need of dentists for reparative work will become less, and that is a consummation devoutly to be wished. I would like to say, before I sit down, that I should like to see dental hygienists as you call them, taught the importance of teeth as vital organs in the human economy, and the children with whom they come in contact made to realize their great importance, since the fact cannot be too much stressed that these organs should be preserved throughout life as far as possible. I say this because there is unquestionably at the present day, and there has been for some time, a very reckless sacrifice of teeth, and often, comparatively speaking, good teeth. Only the other day a lady came to me and said that because of trouble she had in two of her teeth which the dentist did not seem to be able to relieve, she went and had them extracted—had them extracted without a consultation with her own dentist, or physician, and without an *x*-ray examination even. So it goes without regard to teeth and tonsils, and it seems to me if we impress upon the minds of the rising generation the very great physiological importance of these organs, and that they must

not be sacrificed on any account unless they be shown by experts, including x-ray experts, to be actually harmful.

Dr. David H. Bergey: I want to take this opportunity of expressing my appreciation. The subject was ably presented by Dr. Jacquette, and I wish to emphasize what he felt was the very important relation of dental caries to some of the organic diseases. I believe that he is correct in his estimate that the teeth are far more frequently the primary focus in which the infection causing these various organic conditions starts out rather than the tonsils. It is evident that the tonsils are commonly the primary focus where disease originates, because of the large number of carious teeth we sometimes see in pupils' mouths. We can form only one conclusion that any chronic disease from which they may suffer, and usually one finds they do, probably has its primary seat in the diseased teeth. I think that must be self-evident.

Dr. Jacquette (in closing): In answer to the question put by Dr. Anders as to the number of children who are receiving contact with the dental hygienist, I would say that Philadelphia has one dental hygienist to meet the needs of the entire city. In reference to the number of individuals in the country having contact with the dentist, I have seen figures that 15 per cent of the population of the country receive treatment from dentists other than extraction.

Survey of the Olive Industry in America and in Spain, and Relation of that Industry to the Public Health

BY HENRY FIELD SMYTH, M.D.

The olive industry, while dealing with one of the leading fruits of the world and one of the oldest-known food products, has never been considered as of special interest from the public-health standpoint until after the introduction of the olive tree into this country a comparatively few years ago, and the marketing of large quantities of canned ripe or so-called ripe olives prepared in California. In 1919 and in 1920 the medical profession and the public were shocked by reports of a number of outbreaks of botulinus poisoning and sudden death attributed to the eating of canned ripe olives.

Repeated outbreaks, 7 in all,[1] with the alarmingly high mortality of over 70 per cent and a death-list of 36 cases, frightened the public and seriously injured the ripe-olive industry and to a less extent the green-olive industry—many people not discriminating between a sound and a spoiled olive, or between a canned ripe olive and a pickled green olive. Before this time, botulinus was a well-recognized condition, more often attributed, however, to improperly processed home-canned fruits and vegetables than the commercially canned goods. Cured olives have been consumed in large quantities, both as green and as ripe fruit, in Spain from the days of the Roman conquest, and the Spanish green fruit packed in brine has been a generally accepted article of diet in this country for as long as most of us can remember. As far as I know, there has never been a case of fatal illness traced to green olives, here or in Spain, and none to ripe olives until the outbreaks above referred to.

California canners, since then, have spent large sums of money on investigations of olive spoilage and of the chemistry of olive curing. Also a vast amount of work has been done by government, state and teaching laboratories on botulinus cultures and on their toxin production and heat resistance. This has resulted in the adoption of increasingly stringent regulations as to sterilization of canned ripe olives.

Both branches of the olive industry have developed to such an extent that any danger or threatened danger from eating cured olives is necessarily a distinct public-health problem of nation-wide importance. They have both long suffered from occasional spoilage of large or small lots of cured fruit, but except for the botulinus outbreaks, due to eating canned ripe olives, these were never a menace to health. However, the green-olive industry has learned the lesson of the experience of its California competitors, and in a scientific way has begun the study of its branch of the industry, with a view to anticipating any possible dangers, to improving and standardizing curing and packing methods, and improving the flavor and keeping qualities of their product.

Until recently both branches of the olive-curing industry were

conducted in purely rule-of-thumb ways and with no attempt at standardization, but rather with a great show of secrecy and dependence on specially developed processes peculiar to individual plants.

Green-olive curing or pickling is an old-established industry, peculiar to Andalusia in Spain, with many traditions and fixed ideas. I have met one man who has been curing olives for forty years and another middle-aged man curing from trees planted by his grandfather. Spain has almost four million acres in olive groves, but 97 per cent of the fruit is grown for oil. The curing of green olives is carried on only around Seville, and that mostly for the American trade. In 1916, 32,144 tons of green olives were cured and the United States paid Spain $2,173,036 for their share of the crop.[2] The business is steadily growing and there are a number of new curers in the field since then. Spain produces twenty-one varieties of olive trees, but only two kinds are used extensively for green curing, the small Manzanillas, running from 200 to 400 per kilo, and the so-called Queen olive, Gordal or Sevillano, which matures so well immediately around Seville, but nowhere else. All green olives shipped to the United States are cured within ten or fifteen miles of Seville, and are shipped by one line of steamers direct from Seville to New York. The whole olives come in hogsheads of 970 pounds, or 170 gallons, and the pimento-stuffed olives in barrels holding 290 pounds.

Olives picked green, in September and October, are gathered by hand and carried in donkey panniers direct to the curing plant, where as soon as possible they are placed in cement vats and covered with a weak caustic alkali. Soaking for four to eight hours softens the skin and removes the naturally bitter taste of the fresh fruit. When the caustic has penetrated one-quarter to one-half of the distance to the stone it is run off, the fruit washed in two or more changes of water, and then placed in a 10 per cent brine solution made from sea-salt and headed up in the large casks. These are rolled into the sun and left with open bungs. They undergo a prolonged, natural, gassy fermentation, which mellows the fruit and develops the desired flavor and olive-green color.

Froth and surface scum are flushed off daily with fresh brine. At the end of fermentation fruit is sorted to size and stuffed or directly repacked in fresh brine, in which it is held until it is repacked in glass in this country. The keeping quality of such fruit depends on the exclusion of air, the salt content of the brine and the acidity developed by fermentation, and spoilage is due to defects in one or more of these factors. The combination of salt and acid inhibits growth and toxin formation of such organisms as botulinus, and, as far as I was able to learn from Spanish physicians and bacteriologists, botulism and botulinus bacilli are unknown in the Seville area. Although pickled olives cannot be sterilized, yet careful sorting when picking and after fermentation, and again before packing in jars, should ensure none but firm, sound, healthy fruit reaching the consumer—kept sound by air exclusion, salt and acid. Properly cured and packed, green fruit keeps indefinitely, and I have examined a jar packed in 1898 still in good condition.

Ripe olives have been cured and eaten in southern Europe for many years, and imported to the United States for consumption by the foreign element, but they were not generally used here until the development of the ripe-olive canning industry in California. The first olive grove was planted by Spanish priests at San Diego, in 1769, and now there are olive groves in thirty-eight counties. There were 834,939 bearing trees in 1916, and in that year 300,000 six-gallon cases of canned olives were marketed.[3] The California olive is not as firm in texture as the Spanish and does not cure green as well, so they have devoted all their energies to the canning of ripe olives. In spite of scientific studies, conducted since 1916, their product has not yet been standardized, due to trade jealousies and the use of a number of different types of fruit mostly developed in California and requiring differences in treatment. For the finished product there is no standard for color, flavor or texture. Some olives are canned when ripe, but the bulk of so-called ripe California olives are picked for canning when just beginning to turn yellow or light brown, and darkened by oxidation from exposure to air after alkali treatment or by active aëration during

alkali treatment or subsequent washing. A very dilute caustic is used and kept on the olives until it penetrates to the stone; subsequent washing is very thorough. After washing, olives are brined in 5 per cent salt and held for some time, or else processed at once. No fermentation develops, and canned olives are neutral in reaction. Unless thoroughly washed, sterilization at high temperature tends to soften the fruit, and so there is a tendency to under- rather than oversterilize. As the botulinus bacillus is more highly resistant to heat than are many putrefactive spore-formers, this may result in killing the latter, which cause disagreeable odors and tastes in spoiled fruit. The far more dangerous botulinus organisms survive and produce fatal amounts of toxin without necessarily causing the unpleasant tastes, smell and appearance that would prevent many from eating them (though it is surprising what some people will eat in the way of spoiled food). Holding olives in brine before processing, as is often done, may also permit botulinus organisms to enter and multiply in fruit originally sound.

Bacillus botulinus is a strictly anaërobic spore-former, not pathogenic in itself and not producing toxin in the animal body. Growing in dead animal or vegetable substances, it produces a most highly virulent toxin—one of the most virulent toxins we know—which Bronfenbrenner has proved fatal to mice in as small an amount as 3×10^{-21} cc (0.000000000000000000003 cc), an amount little larger than the theoretical size of the toxin molecule.[4] Gastric acidity increases rather than decreases its toxicity. Fortunately this toxin is completely inactivated by thirty minutes' boiling, so that doubtful foods may always be so rendered safe. The spores, however, resist boiling for six hours, and resist 110° C. (230° F.) for ten to twenty minutes. . . . Owing to slowness of heat penetration in cans packed in large sterilizers, present regulations call for processing at 240° F. for forty minutes. Cans so processed should be perfectly safe, but a shorter time or lower temperature may not kill all spores and then toxin will develop in sealed cans and render their contents fatal poison.

We have had seven outbreaks of botulinus poisoning in this

country from eating spoiled canned ripe olives, with 36 fatalities, from 50 to 100 per cent of those eating olives from individual cans dying usually within forty-eight hours. In most instances olives responsible for these cases were visibly spoiled, and by most people would have been considered unpleasant to taste or smell. One such olive tasted, however, may cause serious or even fatal illness. In Memphis, Tenn., in 1920, 7 people tasted olives purchased at a store specializing in the marketing of "reclaimed" goods, and they all died.[5]

In New York City, in 1920, 7 Italians were poisoned—6 of them fatally—from fruit resold by a California canner after being refused by a New York jobber as unfit to eat.[6] In 1919, in Canton, Ohio, 17 persons tasted olives from one can, 14 of them became ill and 7 died, the lowest mortality of any of the outbreaks. An interesting fact about this outbreak, and one other not due to olives, was that several nonfatal cases apparently owed their recovery to free indulgence in alcoholic beverages after eating the olives.[7] Other fatalities from eating ripe olives have occurred in Michigan, Montana,[8] Detroit[9] and California.[8]

Botulinus bacilli have been frequently isolated from the soil in California, and many other parts of the United States and Canada, and also from Belgium, Denmark, England, Netherlands, Switzerland, Hawaii and China. Two distinct strains have been described, designated A and B, giving different toxins and producing unlike antitoxins in immunized animals. Type A predominates in California and was isolated from samples of the olives causing most if not all of the seven outbreaks referred to. Its known prevalence in California will call for constant vigilance among olive and vegetable canners there to ensure thorough sterilization. So far there have been no new cases since the present regulations have been in force.

This year there was a bumper crop of olives in California, and the growers have been shipping green and partially ripe olives all over the country for home canning and publishing receipts in local papers for the same. These receipts give no warning as to the danger of understerilization, and do not mention sterilization

directly. In a subsequent paragraph the statement is made that olives cured and placed in brine may be held in the solution for months and processed at the convenience of the householder, with no definition of processing and no directions as to time or temperature of heating. Home processing is notoriously apt to be inadequate for complete sterilization, and there have been fifty-one outbreaks of botulism in the United States from eating home-canned foods as against thirty-one from commercially canned products. It is sincerely to be hoped that these California grown olives canned in domestic households throughout the country do not become responsible for more cases of botulism, but it would not be at all surprising if they did.

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DISCUSSION

Dr. Alexander C. Abbott: It is a very difficult paper for anyone to discuss who has not some technical knowledge of the subject, but I do not like to see it go without some discussion, because of its importance. There are two or three points to me very interesting. Some of you may wonder why Dr. Smyth has laid such stress on the geographical distribution of the botulinus. There are analogies. The tetanus bacillus is a soil germ, as all are aware, but one may take up many specimens of soil in various places without finding the organism. On the other hand, it may be demonstrable in practically all soils from other localities. It is also of very great importance to understand why poisonings are more fatal in the preserved ripe olives than in those prepared green.

Dr. Smyth: The botulinus poison is stated to be a definite toxin, and will produce antitoxin which is of definite value provided it is used

early. Unfortunately, these cases develop so very rapidly that it is only the last cases developing in a single outbreak that the antitoxin reaches in time. It will absolutely protect laboratory animals and in some instances has apparently furnished protection in outbreaks of the poisoning. There have been more outbreaks of botulinus poisoning from home-canned spinach than from any other single food. I have referred to this in this paper. Most botulinus poisoning in Europe has been from canned meat and fish, and has been from type B.

Dr. David H. Bergey: Did death occur suddenly?

Dr. Smyth: Yes. Most of the cases have developed within forty-eight hours. Some of them within less than twenty-four hours. The toxin seems to take effect on the motor nerve-endings, and the symptoms are those of paralysis very rapidly developing, starting in the limbs and spreading rapidly, affecting the throat and the involuntary muscles.

Dr. Abbott: Have you seen anything of it in your practice, Dr. Anders?

Dr. James M. Anders: I have not.

Dr. Abbott: There has been very little of it in this immediate vicinity?

Dr. Anders: Very.

ANNUAL REPORT OF THE LIBRARY COMMITTEE FOR 1923

Mr. President: In accordance with the ordinances and By-Laws of the College, I herewith submit the following report of the Library Committee for the year 1923:

Total number of volumes in the Library, including the bound volumes and 20,827 unbound "Reports" and "Transactions"	140,517
Number of unbound "Theses" and "Dissertations" .	20,208
Number of unbound pamphlets	139,394

Included in the above are 4,184 volumes known as "Reserves," consisting of second copies of some of the more important periodical publications; also 2,903 volumes more or less incomplete.

The duplicates, which number 11,291 at this date, are not included in the above total.

The following table shows the number of volumes in the various divisions of the Library:

	Bound.	Incomplete and unbound.	Total.
General Library	99,111	2,856	101,967
Lewis Library	13,756	44	13,800
On permanent deposit:			
S. D. Gross Library . .	3,703	3	3,706
Library of the Obstetrical Society of Philadelphia .	217	0	217
			119,690

Received during the year from all sources, 5,376 volumes, 9,236 pamphlets and 9,329 numbers of various periodicals.

Divided as follows:

	Volumes.	Pamphlets.	Journals.
General Library	4,717	9,098	9,281
Lewis Library	18		
S. D. Gross Library	15		
By purchase, General Account .	132		
In exchange	494	138	48
	5,376	9,236	9,329

Accessions (including 40 volumes of "Reserves"): 3,186.

	Volumes.
General Library	3,153
Lewis Library	18
S. D. Gross Library	15
	3,186

Total increase in the number of volumes for the year, 3,186.

Photographs received in response to requests sent out during the past year and voluntary gifts: Fellows, 4. For the War Album (Fellows of the College), 1. Other than Fellows, 78.

Total number of portraits listed, 10,776.

The individual "Donors" for the year ending November 1, 1923, number 448; this represents 1,080 distinct presentations. Each gift is duly acknowledged and properly recorded.

The following list shows individual gifts of more than 25 volumes, and the number of volumes presented by the various publishing houses:

	Volumes.
Dr. E. E. Montgomery	798
Pennsylvania Hospital	200
Mütter Museum	189
Dr. A. A. Eshner	185
Mrs. R. M. Bolenius	164
Dr. Stricker Coles	158
Dr. De Forest P. Willard	131
Dr. W. W. Keen	79
Children's Hospital	72
Dr. F. X. Dercum	64
The Misses Kirkbride	64
Dr. A. W. Watson	64
Miss Margaret G. O'Hara	52
Dr. George W. Norris	41
Dr. Alfred Gordon	33
Dr. A. H. P. Leuf	33
Dr. Bernard Kohn	26
Dr. Lewis H. Adler	25
Dr. W. H. Blakeslee	25
Dr. A. W. Hammer	25
Miss Mary Louise Seltzer	25

From the publishing houses of:

P. Blakiston's Son & Co.	15
F. A. Davis Company	17
Lea & Febiger	16
J. B. Lippincott Company	21
W. B. Saunders Company	48

The Library is indebted for large gifts of pamphlets and unbound periodicals to the following donors:

Dr. L. Pierce Anthony
Dr. A. P. C. Ashhurst
Dr. W. H. Blakeslee
P. Blakiston's Son & Co.
Dr. Charles W. Burr
Dr. Burton Chance
Dr. Edward P. Davis
Dr. W. A. Newman Dorland
Dr. H. M. Fisher
Dr. E. H. Goodman
Dr. Hobart A. Hare
Dr. Addinell Hewson
Dr. C. F. Judson
Dr. W. W. Keen
Dr. Bernard Kohn
Dr. Joseph Leidy
Dr. A. H. P. Leuf
J. B. Lippincott Company
Dr. Edward Martin
Dr. Charles K. Mills
Mrs. J. K. Mitchell
Dr. E. E. Montgomery
Dr. Elliston J. Morris
Dr. B. B. Neubauer
Dr. George W. Norris
Dr. Francis R. Packard
Dr. J. H. W. Rhein
Dr. David Riesman
W. B. Saunders Company
Dr. G. E. de Schweinitz
United States Department of the Interior
United States Public Health Service
Dr. James C. Wilson
Dr. William Zentmayer

938 new publications were added to the Library during the past year, viz: United States, 339; Great Britain and Colonies, 265; France, 108; Germany, 207; Italy, 7; all other countries, 12. Of these new publications, 29 were written or edited by Fellows of the College.

26 volumes were presented by the following authors or editors:

Dr. Robert Abbe	Dr. Charles Janet
Dr. William S. Bainbridge	Dr. William W. Keen
Sir Jagadis Chunder Bose	Dr. James Hendrie Lloyd
Dr. Albert P. Brubaker	Dr. B. B. Vincent Lyon
Dr. Francis X. Dercum	Dr. Charles K. Mills
Dr. Wells P. Eagleton	Dr. James Boyd Neal
Mr. K. G. Falk	Dr. J. A. Nixon
Dr. William A. Fisher	Dr. William J. Robinson
Dr. Hobart A. Hare (Editor)	Sir Humphry Rolleston
Dr. Robert H. Ivy	Dr. Herbert E. Walter
Dr. Chevalier Jackson	Dr. Henry R. Wharton

3 volumes were sent by the publishers at the request of the following authors or editors:

Dr. William W. Keen Dr. B. B. Vincent Lyon
Dr. Howard A. Kelly

Summary of the "Funds:"

	Volumes purchased.	Cost.
Henrietta Rush Fales Baker Fund	72	$364.44
Luther S. Bent Fund	21	58.91
William T. Carter Fund	34	94.28
Gerardus Clarkson Fund	19	76.41
Francis X. Dercum Fund	97	423.61
Louis A. Duhring Fund	46	193.18
John D. Griscom Fund	76	260.12
William F. Jenks Fund	93	370.22
Oliver A. Judson Fund	16	70.25
William V. and John M. Keating Fund	23	38.90
William W. Keen Fund	41	297.69
Library Endowment Fund	297	426.52
Horace Magee Memorial Fund	108	767.37
J. Ewing Mears Fund	57	353.74
Charles K. Mills Fund	3	3.47
Weir Mitchell Fund	57	761.36
John H. Musser Fund	16	32.59
Elizabeth K. Newcomet Fund	27	79.03
William F. Norris Fund	22	418.80
Charles A. Oliver Fund	7	94.18
Philadelphia Medical Society Fund	4	73.30
Lewis Rodman Fund	64	300.29
Douglas Stockton Warren Fund	51	192.49
John F. Weightman Fund	19	157.46
Caspar Wistar Fund	35	291.89
	1,305	$6,200.60

Special Accounts:

	Volumes purchased.	Cost.
Fund for completing files of journals	97	$439.18
Fund for rare and valuable books	40	1,702.67
S. D. Gross Library Account	9	24.43
	146	$2,166.28

George B. Wood Fund—Library supplies, stationery, etc. Expended $248.32.

Morris Longstreth Library Fund—On account of salaries. Expended $893.25.

Catalogue Endowment Fund—On account of salaries. Expended, $37.50.

Louis A. Duhring Fund—General purposes of the Library. Expended, $11,662.00.

Number of volumes bound, 1,266.

Use of the Library: Visitors, 6,113; Fellows, 2,172; total, 8,285.

The Library has been kept open two evenings each week and on the six minor legal holidays, for the same hours and period of time, as during the past year:

Evenings.	Visitors.	Fellows.	Total.
79	736	187	923

Legal holidays.	Visitors.	Fellows.	Total.
6	75	28	103

These figures are included in the total given for the year.

	1923.	1922.	Increase.
Number of books consulted in the Library	25,675	18,918	6,757

The number of books reported as "consulted in the Library" includes only those supplied on demand. Readers have access to the bound volumes of periodicals and works of reference kept on the shelves in the Reading Room, and the Fellows have access to the Book Stacks. There are, therefore, a great many volumes consulted of which no accurate record can be kept.

	1923.	1922.	Increase.
Number of books taken out .	3,204	3,186	18

Use of Study Rooms: Average number of rooms in use throughout the year, 4. Greatest number in use at one time, 5. Daily average of the number of volumes in use in these rooms, 110.

Cataloguing: Works, 1,759; volumes, 1,889; cards written, 7,455; printed cards revised, 3,983.

All books added to the shelves during the past year and 171 of the more important pamphlets have been catalogued and shelf-listed; and all bound volumes, including periodicals, transactions and reports, have been accessioned.

Revision of the Catalogue: 3,771 cards have been revised, rewritten and filed.

3,387 unbound pamphlets and reprints have been subject-headed and arranged alphabetically by subject, and by author under the subject. The binding of pamphlets by subject is still held in abeyance on account of the excessive charges occasioned by labor conditions.

We have listed November 1, 1923, 1,190 current periodical publications, including "Transactions" and "Reports," obtained through the following sources:

	American.	Foreign.
Endowment Funds	97	543
By purchase from General Account . .	17	119
In exchange	57	96
Editors	103	15
Publishers	19	2
In addition, current numbers of periodicals have been received, at stated intervals, through the courtesy of the Editor of the *Therapeutic Gazette*	28	34
	321	809
Reports not included in the above . . .	138	12
	459	821

The actual number of periodicals received during the year, including new subscriptions (2 American and 4 foreign), total 941. This is an increase of 11 over the previous year. We are still carrying our lists intact, and changes are made only when official notification is received from our agents, or from the editors, or publishers, of the journals direct.

The following is a list of the foreign schools of medicine with which we exchanged publications prior to the late war:

University of Amsterdam	University of Königsberg
" Basel	" Lausanne
" Berlin	" Leiden
" Bern	" Leipzig
" Bonn	" Liège
" Breslau	" Lund
" Erlangen	" Marburg
" Geneva	" Rostock
" Giessen	" Strassburg
" Göttingen	" Upsala
" Greifswald	" Utrecht
" Halle	" Würzburg
" Heidelberg	" Zürich
" Kiel	

Faculty of Medicine of Bruxelles
" " Paris
" " Toulouse
" " Yucatan

It was hoped that after the disturbance incident to the war had subsided, all the institutions named would resume their duties; therefore, no revision of the list has been made and our publications have been forwarded regularly. To a great extent our hopes have been realized, as the dissertations, or theses, have been received, with more or less regularity, from twenty-four of the institutions listed above. Conditions resulting from the war are such that it is quite probable modifications of some kind, in the requirements of dissertations from candidates, have been made by many of the universities mentioned in our list.

During the past year we have heard from Amsterdam, Basel, Berlin, Bern, Breslau, Giessen, Halle, Heidelberg, Kiel, Königsberg, Lausanne, Leiden, Liège, Marburg, Strassburg and Utrecht. 2,496 dissertations and 35 theses have been added to our collection.

We have received in cash from the sale of duplicates for the current year ending November 1, 1923: $327.15.

During the past year we have distributed books and journals, on exchange account, to the following:

Boston Medical Library
New York Public Library, New York City

And have received exchanges from:

Boston Medical Library
Mayo Clinic Library, Rochester, Minn.
Medical Library Association, Baltimore, Md.

With the aid received from exchanges and purchases made with funds appropriated for the purpose by the Library Committee, we have been able during the past year to fill in gaps in the files of the following journals:

Archivio di psichiatria, scienze penali ed antropologia criminale, Torino
Bullettino delle scienze mediche, Bologna
Congrès des médecins aliénists et neurologistes de France, Paris
Deutsche militärärztliche Zeitschrift, Berlin
Giornale Italiano delle malattie veneree e della pelle, Milano
Journal of the New York Microscopical Society, New York City
La Pediatria, Napoli
Verhandlungen des vereins für innere Medicin und Kinderheilkunde, Berlin und Leipzig
Zeitschrift für angewandte Psychologie und psychologische Sammelforschung, Leipzig
Zeitschrift für Kinderheilkunde, Berlin

Amount of fines collected from November 1, 1922, to November 1, 1923: $101.85.

Through the courtesy of the Editor, Dr. Francis R. Packard, there was printed in the first number of Volume V of the *Annals of Medical History* a revised list of the Incunabula in the Library of the College. Corrected reprints of this list were obtained, and, under the title of "Catalogue of the Incunabula in the Library of the College of Physicians of Philadelphia [1923]," were distributed to the principal medical libraries in this country and abroad. It is with gratification that the Library Committee can report the completion of this list and its distribution, as it possibly represents the largest collection of Fifteenth Century printed books, relating to medicine, in any but the Government supported libraries of the world. At the same time this Catalogue, as is the case with all printed catalogues of active and growing libraries, was practically imperfect or incomplete as soon as it left the printer's hands, as can be readily verified by referring to the following list of incunabula added during the current year:

Incunabula

[Total number of incunabula at this date, 359 Titles, 341 Volumes]

These fifteenth century books constitute an invaluable collection for the student of early history and foundations of medical science. They have been acquired for the Library without in any way interfering with the purchase of current medical publications—books, journals, etc. The Library Committee fully realize that the first object of the Library is to supply its readers with all the current medical literature. This imperative need having been complied with, it is gratifying to report that, in the course of years, we have been able to increase the extremely valuable collection of rare books, the gift or legacies of former Fellows; so that it is now a practically unique collection of the original editions of the medical classics.

Aegidius Columna s. de Roma, Ord. Eremit. S. August. [Commentum super libros duos posteriorum analyticorum Aristotelis.] Venetiis, Andreas Toresanus de Asula per Simonem de Luere, 1500. [Hain 139.]

Horace Magee Memorial Fund.

Albertus Carrariensis, Joh. Mich. De omnibus ingenii augendae memoriae libellus. Bononiae, Plato de Benedictis, Bononiensis, 1491. (Hain 426.)

Fund for Rare Books.

Aristoteles. Meteorum liber primus cum commentariis Caesani de Thienis. Venetiis, Johannes et Gregorius de Gregoriis, 1491. (Hain-Copinger 1697.)

Fund for Rare Books.

Aristoteles. (Opera lat. cum commentariis Averrois.) Venetiis, Bernardinus Stagninus de Trino, de Monteferrato, 1488. (Hain 1661.)

Contemporary binding, wooden boards covered with stamped leather, with two metal bosses. Fly-leaves from early Thirteenth Century Mss.

William F. Norris Fund.

Aristoteles. (Problemata.) Questiones naturales aristotelis De variis corporum humanorum dispositionibus audientibus valde vsuales.*** Antverpiae, Gerhardus Back. (1500). (Hain 1738.)

William F. Norris Fund.

Aurelius Victor, Sextus (pseudonym. Plinius Primus). De rebus praeclare gestis virorum illustrium romanorum. (Venetiis, Nicolaus Jenson, 1471.) (Hain-Copinger 2131.)

Fund for Rare Books.

Ausonius. (Opera.) Epigrammata. Venetiis, Johannes Tacuinus, 1494. (Hain 2178.)

(*Contemporary Mss. notes.*)

Fund for Rare Books.

Baverrus de Baverris, Johannes. Consilia medicinalia. Bononiae, Plato de Benedictis, 1498. (Hain-Copinger 2712.)

Fund for Rare Books.

Boethius, Anicius Manlius Torquatus Severinus. Opera. 2 Volumes in 1. Venetiis, Johannes et Gregorius de Gregoriis, 1491–1492. (Hain 3353.)

Fund for Rare Books.

Cantor Frisius, Johannes. Prognosticon Anni Mccccxxxviij. (Venetiis, 1488.)

(*Not in Hain.*)

Fund for Rare Books.

Castello, Turan de. Tractatus de balneis.*** [Vicentiae] Johannes de Rheno, 1473. [Hain 4592.]

Only six other works issued from this press. Seven pages of notes in a contemporary hand.

Fund for Rare Books.

Champerius, Symphorianus. Dyalogus singularissimus *** in magicarum artium destructionem. Lugduni, Guillermus Balsarin [circa 1500]. [Copinger 1570.]

Full-page woodcut on reverse of title.

Fund for Rare Books.

Concoregia, Johannes de. Opus de aegritudinibus particularibus. Papiae, Antonius de Carchano, Mediolanensis, 1485. [Hain 5615].

Fund for Rare Books.

Eyb, Albertus de. Ob einem Manne zu nehmen ein ehelich Weib. [Lipsiae, Martinus Landsberg de Herbipoli, circa 1472.] [Hain 6828.]

Fund for Rare Books.

Fasciculus temporum (autore Wernero Rolevinck Carthusian). [Coloniae] Henricus Quentell, Coloniensis, 1480. [Hain 6925.]

Fund for Rare Books.

Gellius, Aulus. Noctes atticae. Venetiis, Andreas Jacobus Catharensis, 1477. [Hain-Copinger 7520.]

Fund for Rare Books.

Gerson, Johannes. Opera. 3 Volumes. Basileae, Nicolaus Kessler, 1489. [Hain 7624.]

Fund for Rare Books.

[Hortus sanitatis.] Moguntiae, Jacobus Meydenbach, Moguntinus, 1491. [Hain-Copinger 8944.]

[*Perfect copy of this rare edition with last blank containing Mss. index by a contemporary hand.*]

Fund for Rare Books.

Hugo Senensis. Consilia medica. Papiae [Leonardus de Gerlis, 1498.] [Reichling 1544.]

Fund for Rare Books.

[Instructiones confessorum.] [Magdeburgi, Albertus Rauenstein et Joachimus Westval, 1483.] [Hain 9248.]

Fund for Rare Books.

Lactantius, Lucius Coelius Firmianus. [Opera.] Venetiis, Vincentius Benalius, 1493. [Hain-Copinger 9816.]

[*Roman type. Only four books known to have been printed at this press, of which only two are in Roman type.*]

John D. Griscom Fund.

Lotharius Diaconus Cardinalis, postea Innocentius III. Papa. Liber de miseria humanae conditionis seu de contemptu mundi. Norimbergae, Fridericus Creusner, 1477. [Hain 10216.]

Fund for Rare Books.

Ptolemaeus Alexandr., Claudius. [Opus quadripartitum.] Venetiis, Octavianus Scotus, Modoetiensis, per Bonetum Locatellum, Presbyter. Bergomensis, 1493. [Hain 13544.]

Fund for Rare Books.

Works of Special Interest

Adams, George. Essay on electricity, explaining the theory and practice of that useful science and the mode of applying it to medical purposes. With an essay on magnetism. 3 Ed. London, Hindmarsh, 1787.

Presented by the Pennsylvania Hospital.

Affo, Ireneo. Saggio di memorie sur la tipografia parmense del secolo XV. Parma, Dalla Stamperia Reale, 1791.

Fund for Rare Books.

Argenterius, Joannes. Opera nunquam excusa, iamdiu desiderata, ac è tenebris in lucem prodita.*** Venetiis, apud Iuntas, 1606.

First edition.

Fund for Rare Books.

de Arles y Andosilla, Martinus. Tractat[us] insignis et exquisitissimus de supertitionibus contra maleficia seu sortilegia que hodie vigent in orbe terrarum.*** Parisius, 1517.

Very rare first edition.

Fund for Rare Books.

Arthy, Elliot. Seaman's medical advocate; or, an attempt to shew that five thousand seamen are, annually, during war, lost to the British nation, in the West-India merchants' service, and on-board ships of War on the West-India station, through the yellow fever.*** London, Richardson, 1798.

Presented by the Pennsylvania Hospital.

Aurelianus, Caelius. Medici vetusti, et in tractanda morborum curatione diligentissimi secta methodici.*** Lvgdvni, Apud Rovillium, 1567.

Rare.

Fund for Rare Books.

Avicenna. Flores Auicenne collecti super quinque canonibus quos edidit in medicina; nec non super decem et nouem libris de an[im]alibus cum canticis. Lugd[uni] Trot, 1514.

Caspar Wistar Fund.

Bacon, Roger. Epistola *** de secretis operibus artis et naturae, et de nullitate magiae, opera Johannis Dee.*** Hamburgi, Froben, 1616.

Henrietta Rush Fales Baker Fund.

Beckett, William. Free and impartial enquiry into the antiquity and efficiency of touching for the cure of the King's evil.*** London, Peele, 1722.

Fund for Rare Books.

de Bezanson. Nouveau traité des fievres, où aprés avoir examiné les differens systemes qui ont paru sur ce sujet, on explique la nature et les causes de ces maladies avec des remedes propers à leur guerison Paris, d'Houry, 1690.

Douglas Stockton Warren Memorial Fund.

Biagini, Luigi. Rapporto storico-medico delle inoculazione ienneriane eseguite in Pistoja. Firenze, Nella stamperia imperiale, 1808.

William T. Carter Fund.

Bourdet [A.]. Soins faciles pour la properté de la bouche, et pour la conservation des dents. Lausanne, Mourer, 1782.

John D. Griscom Fund.

Boyle, Robert. Disquisition about the final causes of natural things; wherein it is inquir'd, whether, and (if at all) with what cautions, a naturalist should admit them? To which are subjoin'd by way of appendix some uncommon observations about vitiated sight. London, Taylor, 1688.

Fund for Rare Books.

Brouzet, N. Essai sur l'éducation médicinale des enfans, et sur leur maladies. Paris, Cavelier, 1754. 2 Volumes.

Henrietta Rush Fales Baker Fund.

Caesalpinus, Andreas. Artis medicae liber VII (de morbis ventris). Romae, Zannetti, 1593.

Rare first edition.

Fund for Rare Books.

Capella, Galeazzo Flavio. L'anthropologia. Venetiae, della heridi d'Aldo Romano & d'Andrea d'Asola, 1533.

Rare first edition.

Fund for Rare Books.

Cardanus, Hieronymus. Opus novum cunctis de sanitate tuenda, ac vita producenda studiosis apprime necessarium; in quatuor libros digestum. Romae, Zanettus, 1580.

First edition.

John D. Griscom Fund.

Caufape, Anicet. Nouvelle explication des fievres et de la gangrene. Avignon, Offray, 1687.

[*First edition.*]

Horace Magee Memorial Fund.

Cesio, Carlo. Cognitione de muscoli del corpo humano per il disegno opera di Carlo Cesio. Roma, Billio [1750].

J. Ewing Mears Fund.

Champerius, Symphorianus. [Libri.] Liber Galeni *** de oculis.*** Speculum medicine Galeni.*** Medicine propugnaculum.*** [Lugduni] 1512.

William F. Norris Fund.

Charleton, Walter. Three anatomic lectures; concerning the motion of the blood through the veins and arteries; the organic structure of the heart; the efficient causes of the heart pulsation.*** London, Kettilby, 1583.

Horace Magee Memorial Fund.

Complete midwife's practice enlarged.*** Second edition corrected by R. C. [London] Brook, 1659.

Presented by Edward B. Krumbhaar, M.D.

Credal, G. F. Traité de quelques maladies de la poitrine, avec leur diagnostic, prognostic et pancement, fondé sur les observations et sur les préceptes des plus habiles médecines dont on rapporte les aphorismes. Paris, Clouiser, 1739.

William F. Norris Fund.

Dupré de Lisle. Traité des maladies de la poitrine, connues sous le nom de phtisie pulmonaire.*** Paris, Costard, 1769.

Fund for Rare Books.

Erziehung, Gebrauch, Lernung, Arztney in züfelliligen und Natürlichen krankheitten aller zahmen dem menschen, gebreuchlichen und geheimenn Thier *** auss Verrone, Plinio, Vergilio, Palladio, etc. Augspurg, Stayner, 1530.

Presented by Mr. Clement B. Newbold.

Faulkner, Sir Arthur Brooke. Treatise on the plague, designed to prove it contagious, from facts, collected during the author's residence in Malta, when visited by that malady in 1813.*** London, Longman, 1820.

Presented by the Pennsylvania Hospital.

Fletcher, J. Differences, cavses, and ivdgements of vrine; according to the best writers thereof, both old and new, summarily collected. London, Legatt, 1641.

Fund for Rare Books.

Ford, Edward. Observations on the disease of the hip joint; to which are added some remarks on white swellings of the knee, the caries of the joint of the wrist and other similar complaints; the whole illustrated by cases, and engravings taken from the diseased parts. London, Dilly, 1794.

Presented by the Pennsylvania Hospital.

Fox, Edward. Formulae medicamentorum selectae; or, select prescriptions of the most eminent physicians, for various diseases incident to the human body. London, Cadell, 1717.

Presented by the Pennsylvania Hospital.

Gataker, Thomas. Essays on medical subjects, originally printed separately; to which is now prefixed an introduction relating to the use of hemlock and corrosive sublimate, and to the application of caustic medicines in cancerous disorders. London, Dodsley, 1764.

Presented by the Pennsylvania Hospital.

Genga, Bernardino. Anatomy improv'd and illustrated with regard to the uses thereof in designing; not only laid down from an examen of the bones and muscles of the human body but also demonstrated and exemplified from the most celebrated antique statues of Rome. Explanation and indexes added by J. M. Lancissi. (sic) London, Senex [1723].

J. Ewing Mears Fund.

[Gesner, Conrad]. The treasure of Euonymus, conteyninge the wonderfull hid secretes of nature, touchinge the most apte formes to prepare and destyl medicines, for the conservation of helth as quintessece, aurum potabile, Hippocras, aromatical wynes, balmes, oyles, perfumes, garnishyng waters, and other manifold excellent confections, whereunto are joyned the formes of sondry apt fornaces, and vessels, required in this art. Translated with great diligence and laboure out of Latin, by Peter Morwyng. London, Daie, 1559.

Henrietta Rush Fales Baker Fund.

Grant, William. An enquiry into the nature, rise, and progress of the fevers most common in London, as they have succeeded each other in the different seasons for the last twenty years; with some observations on the best method of treating them. London, Cadell, 1771.

Presented by the Pennsylvania Hospital.

Grant, William. Some observations on the origin, progress, and method of treating the atrabilious temperament and gout. 3 Volumes in 1. London, Cadell, 1779-1881.

Presented by the Pennsylvania Hospital.

Grevin, Jaques. Deux livres des venins, ausquels il est amplement discouru des bestes venimeuses, theriaques, poisons et contrapoisons. Anvers, Plantin, 1568.

Dedicated to Queen Elizabeth.

*Contains also—de l'Aunay, L. Responce av discours de Maistre Jacques Grevin *** touchant la faculte de l'antimoine,* 1566. *Nikander. Les oevures.**** 1567.

Caspar Wistar Fund.

Guy, Edward. Practical observations on cancers and disorders of the breast, explaining their different appearances and events. To which are added one hundred cases, successfully treated without cutting.*** London, Owen, 1762.

Presented by Edward B. Krumbhaar, M.D.

Heurnius, Joannes. Opera omnia juxta Otthonis Heurnii.*** Postrema ed. 2 Vols. in 1. Lvgdvni, Hvgvetan, 1658.

Henrietta Rush Fales Baker Fund.

Horst, Gregorius sr. Operum medicorum tomus primus, qui continet institutiones medicas, et reliqua scripta theorica huc spectantia cum triplici indice disputationum scil. questionum, ac rerum verborum-[que] magis notabilium, cura Gregorii Horstii. jr. 3 Volumes in 1. Norimbergae, Endter, 1660.

Henrietta Rush Fales Baker Fund.

de Houssemaine, Nicolas. Lentretenement de vie. Comprenant sommairement la guarison de toutes maladies, la maniere de prendre medecine et use de cyrurgie.*** Nouvellement compille par Jehan Goeurot. Paris, Janot [1535].

Horace Magee Memorial Fund.

Joubert, Laur[ent]. Traité du ris, contenant son essance, ses causes, et mervelheus essais, curieusemant recerchés, raisonnés et observés.*** Paris, Chesneau, 1579.

Horace Magee Memorial Fund.

[Kennedy, Peter.] Ophthalmographia; or, A treatise of the eye, in two parts. Part 1. Containing a new and exact description of the eye; as also the theory of the vision considered, with its diseases. Part 2. Containing the signs, causes, and cure of the maladies incident to the eye.*** London, Lintott, 1713.

William F. Norris Fund.

a Lacuna, Andrea. De articulari morbo commentarius.*** Romae, Dorius, 1551.

[*Contains also—Lucian of Samosata. Tragopodagra.****]

Fund for Rare Books.

[de La Faye, Georges.] La chirurgia completa secondo il sistema de'moderni. Trad. dal francese.*** Venezia, Remondini, 1758.

Presented by Mr. Sennaro L. Salerno.

Lambe, William. Reports on the effects of a peculiar regimen on scirrhous tumours and cancerous ulcers. London, M'Creaey, 1809.
Presented by the Pennsylvania Hospital.

Lavater, J[oannes] H[enricus]. Elémens anatomiques d'ostéologie et de myologie à l'usage des peintres et sculpteurs. Traduits de l'allemand par Gauthier de la Peyronie.*** Paris, Tilliard, 1797.
J. Ewing Mears Fund.

Lipscomb, George. Observations on the history and cause of asthma; and a review of "A practical enquiry on disordered respiration" in a letter to Robert Bree, M.D. Birmingham, Belcher, 1800.
Presented by the Pennsylvania Hospital.

Lobera de Avila [Luis]. Bancket der Hofe und Edelleut. Des gesunden lebens Regiment. Von eygenschafft, nutz unnd schedlicheyt alles so Menschlicher speise, tranck, und gebrauch inn Küchen, Keller unnd Apotecken.*** Frankfurt, Egen[off], 1556.
Rare.
Fund for Rare Books.

Magirus, Joannes. Anthropologia; hoc est, Commentarius *** Philipp Melanchthonis libellum de Anima,*** opera Georgii Caufungeri. Francofurti, Nebenii, 1603.
Fund for Rare Books.

de Marian, Dortous. Eloges des académiciens de l'Académie royale des sciences morts dans les années 1741, 1742 et 1743. Paris, Durand, 1766.
Presented by Alfred C. Wood, M.D.

Mazzuchelli, Gian. Maria. Notizie storiche e critiche intorno alla vita di Pietro D'Abano. Venezia, Occhi, 1740.
John D. Griscom Fund.

Merindolus, Antonius. Ars medica in duas partes secta in qua non solum ea explicantur, quae ad medicinam discendam sunt necessaria; sed multa etiam, qua theologos et philosophos recreare valeant continentur.*** Aquis Sextiis, Roize, 1633.
Henrietta Rush Fales Baker Fund.

Nonus, Theophanus. De omnium particularium morborum curatione sic ut febres quoque et tumores praeter naturam complectatur, liber, nunc primum in lucem editus, et summa diligentia conuersus *** per H. Martium. Argentorati, Ribelius, 1568.
Fund for Rare Books.

Pancirolli, Guido. Rerum memorabilium iam olim, deperditarum, et recens atque ingenioses inventarum; Libro duo,*** editi *** per Henricum Salmuth. Volume 1. Ambergae, typ. Forsterianis, 1590.

Presented by Mr. Clement B. Newbold.

Panizza, Ludovicus. De venae sectione in inflammationibus quibuscunque fluxione genitis, per sanguinis missionem curandis. Venetiis, Camocius, 1561.

Lewis Rodman Fund.

Paré, Ambroise. The works of that famous chirurgeon. Translated out of Latin, and compared with the French by Th. Johnson; together with three tractates concerning the veins, arteries, and nerves; exemplified with large anatomical figures. Translated out of Adrianus Spigelius. London, Clark, 1678.

Presented by Lewis H. Adler, M.D.

Paterson, David. Treatise on the scurvy; containing a new, an easy, and effectual method of curing that disease.*** Edinburgh, Manners, 1795.

Presented by the Pennsylvania Hospital.

Philoponus, Joannes. Commentaria in libros de generatione, et corruptione, Aristotelis; ex colloquiis Hammonii Hermae, propriisque eius considerationibus, Hieronymo Bagolino Veronensi interprete. Venetiis, Hieronymus Scotus, 1543.

William F. Jenks Fund.

Planerius Quintianus, Joannes. Febrivm omnivm simplicivm diuisio et compositio ex Galeno, et Auicenna excerpta; et in Arbores, et facilius intelligatur, redacta. Venetiis, apvd Iacobvm Vitalem, 1574.

*Contains also his Dubitationum, et solutionum in III. Galeni de diebus criticis liber unus.****

Louis A. Duhring Fund.

de Planis Campy, David. La chirurgie chimique medicale, ou est traicté amplement de l'origine des maladies et curation d'icelles, auec le discourse de la phlebotomie, où est monstré, en bref les deux temps d'icelle. Plus le traitté des crises. Roven, Cailloüé, 1642.

William W. Keen Fund.

de Planis Campy, David. Discours de la phlebotomie ov est monstré en bref, les deux temps d'icelle.*** Plus un traitté des crises.*** Paris, Perier, 1620.

Engraved title-page, dated 1621.

William W. Keen Fund.

Pott, Percivall. Practical remarks on the hydrocele or watry rupture, and some other diseases of the testicle, it's coats and vessels.*** London, Hitch, 1762.

*Contains also his—Account of a particular kind of rupture.*** 1757.
Observations on that disorder of the cornea of the eye commonly called fistula lachrymalis. 1758.

William W. Keen Fund.

Priscianus, Theodorus. Phaenomenon euporiston liber I, Logicus liber II, Gynaecea ad saluinam liber III; opus nunc primum aeditum [per Sigism. Gelenium]. Basileae, Froben, 1532.

Louis A. Duhring Fund.

Riolan, Jean. Comparatio veteris medicinae cum noua, Hipocraticae cum hermetica, dogmatice cum spagyrica. Parisiis, apud Hadrianum Perier, 1605.

Rare first edition.

Fund for Rare Books.

Riolan, Jean. Opera medica.*** Parisiis, apud Ludovicum Bovllenger, 1641.

Fund for Rare Books.

Rutty, John. Materia medica antiqua et nova, repurgata et illustrata; sive de medicamentorum simplicium officinalium facultatibus tractatus. Rotterodami, Dilly, 1775.

Presented by the Pennsylvania Hospital.

Rutty, William. Treatise of the urinary passages; containing their description, powers, and uses; together with the principal distempers that affect them; in particular the stone of the kidneys and bladder.*** London, Worrall, 1726.

Fund for Rare Books.

Samoilowitz, D[anilo Samoilovitch]. Lettre sur les expériences des frictions galaciales pour la guérison de la peste, et autres maladies putrides. Paris, Clerc, 1781.

In Exchange.

Some thoughts on the relaxation of human bodies, and on the misapplication of the bark in that and some other cases. London, Nicoll, 1783.

Presented by J. Solis-Cohen, M.D.

Tabernaemontanus, Jacobus Theodorum. Neuw vollkommeytlich Kreuterbuch*** aller Gewachs, der Baumen, Stauden und Krautern, so in Teutschen und Welschen Landen auch in Hispanien Ost- und West Indien, oder in der Newen Welt Wachsen.*** 4. Aufl. Franckfurt a. M., 1625. 3 Volumes in 1.

Presented by Mr. Clement B. Newbold.

Tesoro delle gioie; trattato curioso, nel quale si dichiara breuemente la virtù, qualità, e proprietà delle gioie, come perle, gemme, auori, vnicorni, beazari, cocco, malacca, balsami, contraherba, muschio, ambra, zibetto, e delle altre cose più famose, e pregiate da tutti li diligenti scrittori antichi, moderni, Arabi, Greci, Latini, Italiani, sacri, et moderni lodate, stimate, e conosciute saluteuoli e medicinali. Raccolto dall'Academico ardente etereo. Reuisto, e accresciuto dall'Academico casinense inquieto. Dedicato à Monsig. Gio. Battista Ardemanio, etc. Milano, Appresso Gio. Battista Bid[elli], 1619.

William W. Keen Fund.

Theodosius, Jo. Baptista. Medicinales epistolae lxviii. in quibus complures uariaeq; res ad médicinam.*** Basileae, apud N. Episcopium Iuniorem, 1553.

Fund for Rare Books.

Ursachen warumb Gott der Hernns Kranckheit und andere Creutze zuschicket oder widerfaren kesset. Mit Biblischer heyliger Schrifft und dero Exempel oder Historian wol begründet. Item sechs Trostsprüche alles leyden deste williger und gedultiger zu tragen. Nürnberg, Knotrn, 1563.

Fund for Rare Books.

Vergilio, Polidoro. Abridgement of the notable worke of Polidore Vergilo conteinyng the devisers and first finders out as well of artes, ministeries, feactes and civil ordinaunces, as of rites, and ceremonies commo[n]ly used in the church.*** Compendiously gathered by Thomas Langley. London, Grafton, 1551.

Fund for Rare Books.

Waldschmidt, Joh. Jacob. Praxis medicinae rationalis succincta.*** Parisiis, Sumptibus societatis, 1691.

Presented by the Pennsylvania Hospital.

Whately, Thomas. Description of an affection of the tibia induced by fever, with observations on the treatment of this complaint. London, Callow, 1810.

Presented by the Pennsylvania Hospital.

White, Thomas. Treatise on the struma, or scrofula, commonly called the King's evil.*** 3d Ed. London, printed for the author, 1794.

Fund for Rare Books.

Wingate, Edm. An exact abridgement of all statutes in force and use upon the 4th day of January in the year of our Lord 1641-[42]. Faithfully extracted out of the said statutes, from the beginning of Magna Carta; to the said time, and alphabetically digested under apt titles.*** London, Leybourn, 1655.

John D. Griscom Fund.

Wood, William. Mechanical essay upon the heart. In three anatomical lectures. London, Bickerton, 1729.

Gulstonian Lecture.

Fund for Rare Books.

W[right] T[homas]. Succinct philosophical declaration of the nature of clymactericall yeares, occasioned by the death of Queene Elizabeth. London, Thorpe, 1604.

Fund for Rare Books.

Young, Samuel. An attempt at a systematic reform of the modern practice of adhesion; especially in relation to the use and abuse of the thread suture; with a view of its merits comparatively with those of the adhesive strap; in the surgery of wounds; being the substance of a course of lectures intended to have been addressed to students of surgery. London, Mathews, 1808.

Presented by the Pennsylvania Hospital.

Manuscripts

Mansfeld, David. Arzneimittel Lehre privatim vorgetragen nach Arneman. Braunschweig, 1825–1826.

Formerly the property of the donor's grandfather, Jacob Marshall.

Presented by John Marshall, M.D.

Say, Benjamin. Accounts with his patients for attendance and medicine. Ledger. [Phila., 1795–1804.]

Presented by Francis Fisher Kane, Esq.

Other Interesting Additions

A photograph of the painting of Pasteur in his Laboratory.
Presented by Etienne Burnet, M.D.

A cartoon of William Jennings Bryan by King, creator of "Skeeziks," entitled "He Denies His Lineage."
Presented by W. A. Newman Dorland, M.D.

A print of the Pennsylvania College and four cards used by the Pennsylvania College Medical Department.
Presented by Mr. Alfred Darrach through the courtesy of Herbert Fox, M.D.

A medal of the Sixth Congress of the International Society of Surgery, London, 1923.
Presented by W. W. Keen, M.D.

A Viennese medal in lead to commemorate the Cholera Epidemic of 1832.
Presented by Edward B. Krumbhaar, M.D.

A framed diploma of Saint Marylebone Infirmary issued to Alfred Elwyn, March 13, 1829.
Presented by Mrs. John K. Mitchell.

A framed photograph of Professor Arlt (Vienna) and his class (about 1866) containing, among others, the picture of Dr. William F. Norris.
Presented by Dr. George W. Norris.

We have also received during the year a number of interesting and more or less valuable autograph letters.

Use of the Photostat: During the past year there have been furnished to Fellows of the College 840 prints, and to other than Fellows, on application, 255 prints. These outside applications include requests from Detroit, New Orleans, New York, Milwaukee, Philadelphia, Terre Haute (Indiana), Wilmington (Delaware), and China.

The work for the Library has been continued according to the original instructions of the Library Committee; 5,928 large and small prints have been made; 25 volumes of incunabula completed as well as various portraits and manuscripts; also a handsome reproduction of the 1513 edition of Rösslin's "Der Swangerer Frauwen und Hebammen Rosengarten," and through the courtesy of Dr. LeRoy Crummer, of Omaha, Nebraska, an excellent photostat reproduction of the work of Canano, entitled "Musculorum humani corporis picturata dissectio," published about 1541. This book is extremely rare, only four copies have been recorded; and through the courtesy of Dr. Guy Hinsdale, Hot Springs, Virginia, a photostat reproduction of the rare work entitled "Aërologia," by Domenico Panaroli, published at Rome in 1642. Up to the present time there have been made photostat copies of 201 incunabula.

At the October meeting of the College it was officially announced that Dr. E. E. Montgomery would present the sum of five thousand dollars ($5,000.00) to be known as the E. E. Montgomery Library Fund; the income of which is to be used for the purchase of books and journals, including the binding of the latter; devoted to Obstetrics, Gynecology and Abdominal Surgery. One thousand dollars ($1,000.00) of the amount has been paid to the Treasurer.

The total of the Library Endowment Funds at this date is: $358,633.92.

An account of stock has been taken in the usual manner, with the shelf list, and any displacements found arranged in their proper order.

There have been four changes in the Library Staff during the past year; this has caused, in comparatively a slight degree, a decrease in the amount of work accomplished in the revision of the catalogue cards, the subject-heading of pamphlets and reprints and the number of impressions of photostat prints.

The figures given of the number of visitors during the past year are practically the same as the preceding year—a difference of but 11 in favor of 1922; there is an increase in the number of books reported as consulted in the Library of 6,757, and an increase in the number of books taken out, of 18.

In regard to the last item, increase in the number of books taken out, attention should be called to a fact which is possibly generally overlooked, and which readily accounts for the comparatively small number of volumes reported as taken out each year—that is, the use of the Study Rooms by the Fellows of the College, the advantage in the use of these rooms is now pretty generally known and thoroughly appreciated. The figures in the present report show a daily average of 110 volumes in use in these rooms. In the old days, or prior to the erection of the present building, with its ten Study Rooms, 50 per cent or more of the volumes collected in a Study Room by a Fellow, and allowed to remain there, unless asked for, until his work is completed, would have been taken out for use at home.

Although figures of the nature presented above are to some extent indicative of the degree of use made by the medical profession of Philadelphia and its vicinity of the facilities offered by the Library, they, of course, have no bearing on the increase in the resources and capabilities of the Library.

In regard to binding, conditions remain practically the same as last year; while there was an increase of 260 in the number of volumes bound, the prices charged are still excessive, and only the more important periodicals and books were sent to the binders.

In closing it seems advisable, as printed copies of this Report will be distributed to the principal medical libraries of the world and to some of the weekly medical periodicals, to repeat the statement that the Library of the College of Physicians of Philadelphia, in point of the number of volumes, in addition to the value of its special collections, is the second largest medical

library in this country, the library of the Surgeon-General's Office, at Washington, holding first place. Among the large and especially valuable and rare collections of medical works, including those in the possession of the large universities of the world, it ranks among the first.

LIBRARY COMMITTEE,
FRANCIS X. DERCUM, M.D.
Chairman.

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